EXPERIMENTAL OBSERVATIONS ON HYPERTONUS *

1. Chairman's General Introduction

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DR. INGRAM lately reminded us† that it is no longer possible to consider muscle tone and posture separately. Now this was not always so. Muscle tone has been recognized for many years as the small resistance offered to passive stretch by healthy muscle, and as a definite firmness of the normal muscle masses when palpated, in contrast to their flabbiness when atonic. And of course it was recognized that damage to the central nervous system altered muscle tone. One view of the purpose of muscle tone was that it was needed for keeping the body warm. Not until Sherrington's work was it realised that the tone of a single muscle is a meaningless entity unless considered in relation to the pattern of posture distributed among the muscles of the body as a whole. Unlike our remote ancestors, we are not supported by water. We are, instead, held in an infinite variety of attitudes by the interaction of gravity and muscular forces. The transition from one of these attitudes to the next is movement. The head, in an animal which moves, as Sherrington pointed out, usually forwards into the environment, occupies a key position in the maintenance of attitudes. It is the function of the trunk and limbs to hold the head erect. The head, as it were, leads the rest of the body, or as Sherrington picturesquely termed it, 'the motor train,' in its wake as it is moved through space. The contrast between normal posture and its absence he most strikingly illustrated by the state of a boxer immediately before and immediately after receiving a knockout blow. It 'reduces in a moment a vigorous athlete to an unstrung bulk of flesh whose weight alone determines its attitude, if indeed a reactionless mass can be described as possessing attitude at all.'

Now for Sherrington the essential basis of posture was the tonic stretch reflex. Receptor organs in stretched muscles excite the ventral horn cells innervating those muscles, causing the development of tension, that is 'muscle tone.' The differential distribution of tone among the musculature as a whole, that is to say, the prevailing posture, was considered to be due to the differential distribution of background excitatory and inhibitory states in the ventral horns of the spinal cord. Some of the local stretch reflexes would be reinforced, others played down. Complex systems of interneurones in the cord and brainstem are the agents immediately responsible for thus distributing the postural contraction. Through these interneuronal systems, impulses from the segmental receptors, from the neck, from the labyrinths and from the eyes, determine, ultimately through the local stretch reflexes, the infinite range of attitudes of which we are capable.

*Papers read at the N.S.S. International Study Group on Child Neurology and Cerebral Palsy, Oxford, Sept., 1958.

†See Dr. T. T. S. Ingram's paper on "Muscle Tone and Posture in Infancy" in No. 5 of this Bulletin, p. 6.

Through all this ran the idea that posture and movement utilized a single motor apparatus. Old notions of a separate nervous control of muscle tone —e.g., by the sympathetic system—were swept away. It was, however, known that different muscles contracted at different speeds, and that the slow muscles were mainly to be found in the antigravity groupings. New research has shown that the motor apparatus is more complex than was once supposed. First came the discovery of the small ('gamma,' 'fusimotor,') motor nerve cells in the ventral horn which excite the receptor organs in the muscles, making them generate stretch reflexes. Next came the discovery that the main ventral horn cells are of two kinds, differing in their reflex reaction to stretch of their muscles. About half of them discharge impulses only at the beginning of stretch ('phasic' motoneurones). The remainder discharge throughout the duration of stretch, or can be made to do so by repeated stretching of the muscle—in Granit's phrase, 'a kind of spastic exaggeration of the response.' Many of these 'tonic' motoneurones are connected to slow muscles. In baby kittens all the muscles are slow, and during maturation some of them become fast. Buller, Eccles and Eccles have discovered that this differentiation is controlled from the ventral horn cells.

Thus the interneuronal systems which control our attitudes and movements may influence the local stretch reflexes by modifying the activity of three types of motor nerve cells, and not, as at first supposed, of only one. Further investigation along these lines is throwing new light on the genesis of spastic states, as you will presently hear from my colleagues.

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2. Hypertonus and the Gamma Motoneurones

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HYPERTONUS is produced by an excessive discharge of the motoneurones supplying the spastic muscles. These motoneurones are of two kinds, the alpha motoneurones supplying the main muscle fibres, and the gamma motoneurones supplying the specialised muscle fibres within the muscle spindles. Activity of the gamma motoneurones does not directly produce tension in the muscle but increases the proprioceptive discharge of the muscle spindles, and this may cause contraction of the muscle by reflex excitation of the alpha motoneurones. The behaviour of the gamma motoneurones might, therefore, be expected to be an important factor in the development of hypertonus (reviewed by Granit 1955).

The role of the gamma motoneurones in the hypertonus of the decerebrate cat has been investigated by selectively paralysing their nerve fibres with procaine, applied locally to the muscle nerve (Matthews and Rushworth 1957). Their paralysis causes the stretch reflex of the muscle studied to become smaller or to disappear, even though the alpha motor fibres and the large afferent fibres which form the direct stretch reflex pathway remain intact. The tendon jerk is similarly diminished. The hypertonus has also been studied by plotting the tension reflexly developed in the muscle against the extension applied to it (Matthews

1958). When the gamma fibres are paralysed the tension-extension curve of the reflex is altered so that a larger extension than before is required to excite any particular degree of reflex contraction. Sometimes the reflex contraction of the muscle disappears at the shortest lengths of the muscle studied, though reappearing at greater lengths of the muscle. The form of the tension-extension relation however alters little when the gamma fibres are paralysed. Thus in decerebrate cats the activity of the gamma motoneurones affects the "threshold" rather than the "stiffness" of the hypertonus.

It may be concluded that in the decerebrate cat the level of activity of the gamma motoneurones is important in determining the degree of hypertonus. It is also probable that in some cases overactivity of the gamma motoneurones is a major cause in the development of the hypertonus. It is important to note, however, that a decrease in the hypertonus when the gamma fibres are paralysed does not show that the gamma motoneurones were abnormally active. It is possible that a normal amount of gamma motoneurone activity may produce or accentuate hypertonus because of some defect elsewhere in the spinal reflex mechanism. Certainly hyperactivity of the gamma motoneurones is not the sole cause of hypertonus, for the stretch reflex may persist when the gamma fibres are paralysed, and gamma overactivity may exist without spasticity (Granit 1955).

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——— and Rushworth, G. (1957) "The selective effect of procaine on the stretch reflex and tendon-jerk of soleus muscle when applied to its nerve." *Ibid.*, **135**, 245-262.

3. The Nature of the Functional Disorder in the Hypertonic States

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BEFORE 1898, the physiology of muscle tonus had already been extensively investigated in continental laboratories, which had produced a wealth of data and an overabundance of confused thinking, largely due to a lack of uniformity of definition. In that year, Sherrington clarified the subject when he published his experiments on decerebrate rigidity in animals — a state of muscular rigidity which ensues when the midbrain is separated from the forebrain at about the intercollicular level. There is a resistance to passive displacement of a limb which is plastic to some extent but building up to a peak until at some point the resistance rather suddenly melts—the lengthening or clasp-knife reaction. It is present in both flexor and extensor muscles and is greatest in those muscles which oppose gravity. The decerebrate animal thus assumes a posture which is a caricature of standing

—in other words, there is an exaggeration of the normal postural reactions of the muscles. Denervation of the skin had little, if any, effect in modifying the rigidity and an already rigid muscle remained so when its neighbours were denervated. Appropriate destruction of the dorsal roots, however, abolished the rigidity of only those muscles which were so de-afferentated. In this way, decerebrate rigidity was shown to be a proprioceptive reflex with its receptor organs lying within or near the effector muscle itself.

The resemblance of decerebrate rigidity in animals to spasticity in man was quickly appreciated as was the feasability of dorsal root section for the treatment of spastic muscles. However, with regard to the effects of de-afferentation on decerebrate rigidity, Sherrington had made some very cautious statements. He says, "The abolition of rigidity is long lasting after section of dorsal roots, i.e. it lasts several hours." And again, "The rigidity develops imperfectly or not at all when the afferent roots have been severed a number of days prior to carrying out the operation of decerebration." In these statements some of us may glimpse a preview of two types of rigidity, one dependent on dorsal root integrity, the other not.

Nevertheless the similarity of decerebrate rigidity and spasticity was further emphasised by Foerster in Germany who noted the effects of a capsular haemorrhage in two patients with tabes dorsalis. One of these patients had involvement of lumbar dorsal columns and roots and following his stroke he was hemiplegic with well-marked spasticity in the upper limb but the lower limb remained flaccid. The second patient had more extensive tabes affecting both lumbar and cervical dorsal roots and posterior columns and following his stroke both hemiplegic limbs were completely flaccid. From these cases Foerster was convinced of the rationale of the operative approach of partial dorsal root section for the relief of spasticity, and in 1911 he published his experiences with this method. At that time he had operated on 62 patients irrespective of the aetiology of the spasticity, but mostly children with Little's disease. He spoke enthusiastically about the results of the operation and illustrated the freedom of leg movement and walking in children who had previously been too spastic to move. Hay Groves in England confirmed Foerster's findings on a smaller number of patients. Two years later (1913) at a meeting of the International Medical Congress in London, Foerster reaffirmed the success of the operation but stated that the spasticity tended to return with partial resection of dorsal roots though it did not return to the previous severity and there was little regression of acquired mobility.

Foerster's operation never became an established therapeutic measure, possibly due to the severity of the operative procedure, especially in the earlier days of neurosurgery, and possibly due to the reluctance to destroy all modalities of sensation from superficial as well as deep structures of segments of a limb. Nevertheless a full critical appraisal of the procedure is still sadly lacking.

In 1919 Liljestrand and Magnus described the effects of injecting procaine into the muscles of decerebrate cats. The rigidity of the muscles rapidly disappeared though the procainised muscle could still be stirred into full activity by stimulating the motor nerves. Walshe in 1924 injected procaine near the motor point of rigid muscle in patients with Parkinson's disease and found that the rigidity was abolished leaving motor power and the tremor unaffected. These experiments were taken to show that procaine selectively blocks sensory fibres without affecting motor fibres and in this way was analogous to the older work on de-afferentation.

Previous speakers have reviewed for us the evolution of modern ideas about the stretch reflex and particularly how the receptor organ for this reflex, the annulospiral ending of the muscle spindle, is itself under the control of gamma moto-

neurones which alter its sensitivity appropriately preceding muscular activity. We were able to show that the rigidity of the ordinary decerebrate cat is highly dependent on the gamma fibres to the muscle spindle. These small motor nerve fibres are more sensitive to procaine than either large sensory or large motor fibres, and when they are blocked by procaine then the exaggerated stretch reflexes regress leaving the muscle flaccid but yet responsive to motor activation. This type of rigidity—gamma rigidity—is probably due to overactivity of gamma motoneurones which thus oversensitise the annulospiral ending of the muscle spindle to the slightest stretch.

The rigidity of the *anaemic* type of decerebrate cat* by contrast is not abolished when dorsal roots are cut and it is only reduced by procaine when alpha motor fibres (and thus movement) are also blocked. This type of rigidity appears to be maintained by a constant stream of activity along alpha-motor fibres and is largely independent of gamma motoneurones.

It seemed reasonable to assume that the spasticity, rigidity or dystonia of human nervous diseases might resemble either the alpha-rigidity, the gamma-rigidity or a mixture of both.

To test this hypothesis patients with rigidity, or spasticity or dystonia have had procaine injected either around a major nerve of an affected limb or directly into a muscle group. The resistance of the muscle to passive stretch has been frequently tested, the tendon jerk and the maximal voluntary power of contraction. Wherever possible the muscle responses have been recorded mechanically or electrically but clinical appraisal has also been employed to a large extent. 31 patients have been studied, 14 had Parkinsonian rigidity (8 post-encephalitic) 12 were spastic, 3 dystonic and 1 was recovering from tetanus.

Walshe's observations (1924) that Parkinsonian rigidity is abolished by procaine before the motor power or the tremor is affected, has been constantly confirmed. There is then no doubt that Parkinsonian rigidity is the gamma-type—that is, highly dependent on gamma-fibres and probably due to overactivity of gamma-motoneurones.

The spastic cases examined have had varying aetiology. Many cases with spasticity are unsuitable for this method of study which requires a maximal voluntary movement of a muscle group as a test for the integrity of alpha-motor fibres. The muscle group usually employed is the posterior tibial group, and the response to passive stretch (recorded electromyographically or assessed clinically), the tendon jerk, clonus (where present) and maximal voluntary plantar flexion have been followed during a slow, progressive sciatic nerve block with dilute procaine.

A typical record in a patient with a familial spastic paraparesis with concentric needle electrodes inserted into both soleus and gastroenemius muscles showed the response—clonic contractions with the rhythmic beating of motor units, which can be interrupted by ankle jerks. When procaine was infiltrated around the sciatic nerve on the same side, by 5 minutes later the response to stretch was little modified, but 10 minutes later the stretch reflex of soleus had completely disappeared and that of gastrocnemius had become very brief and simple. The tendon jerk of both soleus and gastrocnemius was also small; 12 minutes later, the stretch reflex of both soleus and gastrocnemius had been abolished whereas maximal voluntary plantar flexion was still unaffected. The spasticity in this case was therefore highly dependent on gamma fibres.

A myographic record from another case of spastic paraparesis showed voluntary plantar flexion and ankle-jerks. Infiltration of xylocaine around the sciatic nerve

*This type of experimental animal is prepared by tying both carotids and basilar arteries, resulting in a gross infarct of the brain (and anterior part of the cerebellum) as far back as the pons. The classical decerebrate animal was made by sucking away the whole of the brain in front of the midbrain and sparing the cerebellum.—Ed. C.P.B.

resulted in the abolition of clonus and the ankle jerk but his voluntary movement was not decreased—if anything the amplitude increased. A similar phenomenon occurred in a man with a spastic tetraparesis (probably an early moto-neurone disease), and it has been the usual experience to see an abolition of spasticity and of clonus very soon after the procaine infiltration at a time when motor power was unaffected by the procaine, and indeed in most experiments the amount of procaine injected was often insufficient to block many large motor fibres.

Only two cases in which the spasticity was not dependent on the gamma fibres have been seen. Both these cases had vascular lesions of the brainstem. The spasticity was not abolished by procaine but was reduced only as the voluntary plantar flexion was reduced. The tendon jerk likewise diminished as the motor power and was not abolished by the procaine. This is a clear example of maintenance of spasticity by alpha-motor fibres alone.

Finally, the three dystonic patients (with Wilson's disease) and the one case of tetanus all showed the dissociation of their stretch responses and motor power by procaine and were thus examples of gamma-maintained hypertonic states. This was somewhat disappointing, for at the outset one had hoped that spasticity, rigidity and dystonia might clearly fall into the three groups of alpha-dependent, gamma-dependent or a mixture of the two. It now appears that the majority of the hypertonic states require the integrity of gamma-fibres and quite possibly are caused by a constant bombardment from the gamma-motoneurones.

It would seem therefore that, in the so-called hypertonic state, the disorder of function at the spinal level must to a large extent be blamed on the gamma-motoneurones which we know to be important in normal muscular co-ordination. Still it is these motoneurones which are known to fire first in a number of reflexes, and the intensity of the subsequent muscle-spindle discharge ultimately determines the amount of muscular contraction.

If we now have more idea how the hypertonic states are maintained at the spinal level, we have to admit at least a partial ignorance as to why motoneurones should be more active after damage to certain parts of the C.N.S. The theory that hypertonic states are due to interruption of an inhibitory pathway from the brain which normally keeps the postural reactions damped down is highly unsatisfactory, as no such inhibitory pathway has been demonstrated with conviction. Studies on the functions of the cerebral cortex will eventually clear up gamma-maintained hypertonic states. moment it is not easy to link them with the intricate studies on the spinal reflexes except in broad outline. Denny-Brown regards the normal function of the cerebral cortex as a balance of positive and negative behavioural reactions in response to the environment through exteroceptors (visual and tactile particularly). The positive behaviour is exploratory in type (the positive supporting reaction in the lower limb, grasping in the upper limb, and sucking), and the negative behaviour is an avoiding or withdrawal type. Cortical damage alters the balance between these two reactions, so that one may be released and exaggerated, by the other being taken away. During the maturation of the central nervous system there is a prominence of one of these reactions until those parts of the nervous system responsible for the counterpart have fully developed and there is integration of these reactions to a natural balance. In lesions of the frontal lobe, sparing the motor cortex, in man or experimental monkeys, it is found that motor function has disintegrated to an exploratory type of reaction—to tactile stimuli in the hand an instinctive grasp reflex of orientation of the hand, palpation and then grasping; with a moving stimulus across the palm on to the fingers, a true grasp reflex. We ourselves have shown that this reflex is triggered by the

cutaneous stimulus, which in turn activates gamma motoneurones in the spinal cord, and the stretching of the fingers by the stimulus then finds an overactive stretch reflex which, in turn, causes strong, long-continued finger closure (grasping). Stretching the fingers alone without the preliminary moving stimulus in the palm will not elicit the reflex and there is little or no resistance to the stretch. When the motor cortex is also damaged these behavioural reactions to tactile stimuli are lost and spasticity alone is present. The small repertoire of movement remaining is all under the control of proprioceptive stimulation. Nevertheless a patient (or experimental monkey) can learn to utilise some of the proprioceptive reflexes and their synergistic effects. For example, shoulder traction in a spastic arm also produces some finger flexion, so that grasping an object can be initiated by standing some little distance away, projecting the arm and hooking the fingers round the object. The stretch reflex of the finger flexors then completes the act of finger closure.

Avoiding reactions are released by parietal lesions, and, according to Twitchell in Boston, may be seen in many cases of cerebral palsy. He has found, for example, that when a child with infantile hemiplegia attempts to pick up an object, the tactile contact causes reflex avoiding of the fingers (extension), so that grasping is very difficult if not impossible. This involuntary avoiding reaction in the hand accounts for much of the upper-limb disability.

The movement disorder of patients with tremor or with athetosis is due to a conflict of exploratory and avoiding reactions in response to visual and tactile stimuli, and these may be modified by appropriate proprioceptive stimulation. For example, head and neck turning may convert the athetoid arm into a rigid, stable structure so that objects can be picked up in the hand. Probably the various operations on the brain that have been devised to treat athetosis and the other involuntary movement disorders, destroy the conflict of avoiding and exploratory reactions and the result may be a possibly more desirable dominance of one or the other of these types of behaviour. It is my belief that observation of the reflex potentialities of patients with cerebral palsy, in response to visual, tactile and proprioceptive stimuli will not only help us to know more about the nature of the defect in cerebral palsy but will also be most valuable in teaching these patients how to make the best use of their limbs.

" . . . no-one has ever claimed that the democratic process produces the *right* answer. It only claims to produce an answer acceptable to most people.

. . . we hear a lot about human and international cooperation. Taken as a term, "international cooperation" provokes either cynical indifference or religious mania . . . International cooperation really works only if it is based on a number of obvious and concrete factors. Some people may believe that international cooperation and therefore goodwill can be achieved by some supernational and all-embracing agency. This may happen ultimately, but in the meantime it seems to me that the best chance of achieving some kind of universal understanding is for all similar professional organisations throughout the world to fashion the closest relations between each other. In this way general international understanding will grow out of national and international professional organisations. In this the medical profession has led the way . . . "—THE DUKE OF EDINBURGH, PRESIDENTIAL ADDRESS TO THE BRITISH MEDICAL ASSOCIATION, October 28, 1959. *Brit. med. J.* 1959, ii, 839.

CLINICAL RECOGNITION AND DIFFERENTIATION OF HYPERTONUS*

1. The Orthopædic Surgeon's Outlook

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THE orthopaedic surgeon, as a clinician, could define muscle tone as that sensation of fine resistance, imparted to the hands of the examiner, as a joint is put through a full or more limited range of passive movement. Friction within the joint, since it is a constant factor, is ignored.

Basmajian claims that two factors are responsible for general muscle tone (a) the passive elasticity or turgor of the muscular (and fibrous) tissues and (b) the active (although not continuous) contraction of the muscle in response to the reaction of the nervous system to stimuli. Of these two, the second is the more important.

Thus disease, damage, defect or stimulation of the nervous system may abolish, increase or vary muscle tone within wide limits, the variations being classified clinically by the degree or form of resistance offered by the affected muscles to the passive movement of the related joint. A full clinical appreciation of the characteristic features of this resistance in the spastic, tension athetoid and rigidity case can be gained only by varying the force as well as the speed with which the manipulations are performed, by changing the position of the patient, and by repeating examinations on several subsequent days.

In the spastic form of cerebral palsy the hypertonicity of the muscles is due to an exaggeration of the spinal stretch-reflex. Since a continuation of this state of affairs over a prolonged period may progress to contractural deformity, in the correction of which orthopaedic procedures may become necessary, early diagnosis and early treatment is a matter of supreme importance. The stretch-reflex is probably *the* characteristic feature whereby the hypertonus of spasticity is differentiated from that occurring in other forms of cerebral palsy. It is elicited by a sudden passive movement either in extension or in flexion, never in both, usually of the elbow or knee joint. The mechanical stimulus of handling and moving the limb gives rise to a progressive increase in tone of the muscle being stretched, with sudden arrest in the excursion of the movement at a point which is remarkably constant, and might be described conveniently as the threshold of the stretch-reflex[1]. This abrupt but rather soft blocking to further movement conveys a characteristic sensation to the hands of the examiner not unlike that of

*Papers read at the N.S.S. International Study Group, Oxford, September 1958.

1. Electromyography of muscles during stretch, showing that the stretch reflex is elicited in these patients by the slightest elongation of a muscle; animal experimentation suggests that elongation of only a few thousands of a millimetre may be sufficient to elicit a stretch reflex. The curve relating stretch reflex to the amount of elongation of the muscle is almost a rectangular hyperbola, so that at relatively short lengths the tension developed by the muscle is not very large until a certain length is reached when the tension becomes maximum almost at once. It is this sudden rise to maximum tension that Mr. Pollock likes to call the threshold for the stretch reflex. It is rather the threshold of the "build-up" of the exaggerated stretch reflex which makes up spasticity.—G. Rushworth, Recorder.

a hydraulic brake in sudden action. It would appear that the turgor and contractility of the muscle fibres are rapidly and progressively built up to a peak, reaching their acmé with the arrest of further stretching and movement. It might be likened to a rapidly rising graph. A repetition of these sudden flexion or extension movements will produce similar results, but if the manoeuvre is performed slowly and steadily, full flexion or full extension of the joint will occur, without evidence of muscle contraction. Advantage is taken of this feature by the physiotherapist to obtain muscle relaxation and a full range of passive movement. She has "worked through" the stretch-reflex, and is now ready to encourage voluntary activity by the patient.

The Tension Athetoid[2]

The resistance to stretching noted in the muscles of the tension athetoid bears no true resemblance clinically to that observed in the spastic or to that described as rigidity, although to the inexperienced it may suggest the one or the other.

On inspection, the tension athetoid patient may show a quite remarkable degree of muscular development, and on manipulation of the elbows or knees it will be realised quickly that the strength of the limbs is in keeping with their physical appearance—a point only too well appreciated by the parents during the application of braces. Once co-operation has been obtained flexion or extension of a joint by the surgeon will demonstrate quickly that the muscle fibres stretch with notably less elasticity, and the point of final stretch is neither constant nor is it reached with the same sudden finality which characterises the stretch-reflex of the spastic case, rather is there a gradual increase in the "braking" resistance until movement ceases. Variations in the abruptness, speed or strength with which the passive stretching of the muscle is performed does not allow of that full range of joint movement, obtained in the spastic, nor does it provide the dough-like "give" of rigidity. Where there is marked stiffness of the wrist or fingers vigorous "shaking" of the forearm, as demonstrated by Phelps, will result in an astonishing degree of temporary relaxation so that previously tight fingers and wrist joint can be now fully and easily extended. Such a degree of mechanical stimulation would increase the tension of the spastic but has little or no effect on the stiffness of the rigidity case.

In the case of the choreo-athetoid it is the involuntary, incoordinate movements which are the clinically significant features diagnostically, and not the vagaries of muscle tone.

Rigidity

Rigidity, of which three varieties have been described by Phelps, is an uncommon form of cerebral palsy. In my own practice it has been diagnosed on only 17 occasions in the last 482 cases examined. It is, however, a definite clinical entity which can be classified as a "lead-pipe", "cogwheel" or intermittent form depending upon the manner in which the affected muscles stretch and the sensation which is thus imparted to the hands of the examiner.

This form of hypertonus or plasticity can be appreciated only on slow passive movements and can be recognised on flexion or, more usually, on extension movements of the elbow or knee joints, whereas in spasticity the stretch-reflex is elicited on movement in one direction only. Lead-pipe rigidity is, in my experience, the commonest type and, as the name suggests, the muscles give way in a slow sticky manner like toffee, or as a lead-pipe might bend under slow, steady and firm pressure. The sensation is quite characteristic and when once appreciated is readily recognised thereafter.

The cogwheel variety is equally distinctive, the joint extending under pressure

2. Many neurologists would refer to this as Dystonia (either in flexion or in extension)—G. Rushworth.

in a rather slow and staccato manner which well merits the descriptive term—cogwheel.

The term intermittent is self-descriptive in that the increased muscle resistance to stretching, usually of the lead-pipe variety, is present and well defined at one examination, absent at the next, but may again be easily recognised at a third.

Although these three forms of increased tension bear to each other a certain superficial resemblance, experience gained from frequent handling and manipulation of the limbs of these patients will permit of a differentiation of the one from the other —a clinical feature which in association with the history and neurological findings is an important aid to diagnosis and clinical classification.

A word of warning is perhaps necessary at this point, since recognition of these distinctive characteristics may be made difficult by the patient's reaction to his examiner, or to the circumstances of the investigation. It has been shown that rest and voluntary relaxation can abolish neuromotor activity in the muscles of the normal individual and in those of many spastic patients also. Certain patients accustomed to investigations of this type can produce a state of neuromotor inactivity bordering on the atony of a lower motorneuron lesion which may baffle the diagnostic ability of the very elect. Such an extreme degree of voluntary relaxation is unusual in cerebral palsy; nevertheless long experience of these cases has shown from time to time a certain degree of variability of muscle tone, which may result from treatment, from the mechanical stimulation inseparable from a physical examination, or from emotional conflicts within the family circle. Because of these therapeutic, mechanical, personal or environmentally induced variations in muscle tone and hypertonus, a final clinical assessment should be made not after one, but only after several careful clinical examinations.

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2. Polymyographic Studies of Voluntary and Involuntary Movements

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One of the main difficulties in assessing a motor disability is the description of its parameters. In fact the severity of a motor weakness can be measured in a variety of ways but it is probable that the data obtained by "clinical observation" may vary from one observer to the other with regard to the same patient. Moreover, some variation might be expected even from the same observer examining the same patient on separate occasions, particularly if the time interval is of months or years.

The present contribution is based on some work in progress on a group of young patients suffering from a variety of motor disabilities defined under the vague term of "cerebral palsy", and on a group of normal controls. A method has been devised to display in a graphic form the extent, distribution and relative time sequence of the activity of both

agonist and antagonist muscles of the arms and thighs recorded simultaneously on a portable 8 channel pen recording apparatus. Small silver disc electrodes were stuck to the skin of the anterior and posterior surface of both arms and both thighs by means of collodion. Two electrodes were placed on each of the following muscles: biceps, triceps, quadriceps and hamstring with a total of 16 electrodes with an additional earthing point.

The electrical activity of each muscle was recorded, together with that of all the others during both voluntary and involuntary movements and a simple routine was worked out suitable for both severely and slightly handicapped patients. The appearance of muscle activity in the proximal parts of the limbs was different between normal controls and patients. Moreover there was a considerable variability from one patient to the other and in the same patient from minute to minute. However, some basic patterns appeared to emerge and it was possible to study later the graphs and recognise both quantitative and qualitative differences in various patients. In contrast with the normal controls in most patients the relationship between agonists and antagonists in the same limb appeared altered. Often complex patterns of activity in more than one limb were seen, even in the absence of any recognisable movement.

In patients with spasticity without involuntary movements it was possible to assess the presence of an increased electrical activity in the various groups of muscles examined in all the four limbs during a voluntary movement that appeared to occur only in one limb. In patients with involuntary movements (choreo-athetosis) there was a much greater variability of these patterns of activity from the four limbs, both in relation to the involuntary and to the voluntary movements. Full voluntary relaxation as shown by the disappearance of muscle action potentials (flattening of the record) was much more easily achieved by patients with "spasticity" alone (whether two or four limbs were involved), than by patients with choreo-athetosis also. In the majority of patients examined who showed involuntary movements there was also some degree of clinical "spasticity". In response to a sudden noise, provided the stimulus was unexpected, a startle reaction of abnormal kind was seen in the majority of patients, and in addition to movements in the arms there was usually also increased activity in the quadriceps and to a lesser extent in the hamstring muscles. During passive movements there was usually a greater amount of activity in the myogram of the patients than in that of the controls, and more so in one direction than in the other of the passive movements. This phenomenon was more obvious with some angular speeds than others and Dr. Tardieu will show later this particular point with his excellent technique.

It appears from these preliminary observations that a display of the electrical activity of various groups of muscles might be of value as a permanent record of a particular type and degree of motor integration.

As an addition to the basic clinical observation, the simultaneous myograms from the proximal muscles of the four limbs with an eight channel display will offer a measure on paper of both the quality and the quantity of a given motor disability of the kind seen in patients with so-called cerebral palsy. A comparison of the data obtained from a given patient at intervals even of years might become possible on objective criteria.

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3. The Recording Dynamometer in the Assessment of Motor Function

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ONE of the problems which confront the clinician in dealing with a disorder of motor function is that of quantitative assessment. Another is to devise some method of recording the characteristics which distinguish abnormal from normal movements.

Apart from these requirements some recording methods give results which are of considerable theoretical significance and Sir Gordon Holmes' studies on cerebellar disorders are in this class.

The third requirement is that any device shall be suitable for use in a wide variety of patients and shall be simple, portable, and robust. This approach is rather different from that of the physiologist who often has to gain the maximum information from each of a relatively small number of patients. Our aim is to obtain selected information from as many members of a group as possible.

As far as technique and design are concerned it is easier to measure the strength of the hand grip rather than any more complex function of movement.

The dynamometer designed by Dr. Ritchie Russell and Dr. Schuster has proved to be most effective in meeting the requirements outlined above.

The hand grips of the instrument are connected to a lever which is restrained by a Salters spring balance. These balances are widely available and cover the weight range of 4-30 lbs. in 4 steps. The lever for each hand also carries a ball point pen which moves on the transverse axis of 6 inch recording paper. The paper is driven by clockwork and there are two speeds. (*Examples of recordings were shown from cases of chorea and choreo-athetosis.*) In these conditions it is the interruption of effort rather than any reduction in the static tension developed which is such a striking feature.

Attempts are in progress to use pursuit meters in the characterization of motor disorders. Fundamentally these devices consist of a control knob for each hand which enables the subject to move a pointer and thus attempt to follow the course of another pointer whose excursion is predetermined by the design of the machine.

This work is in its early stages, and the records are rather difficult to interpret. However the approach appears to be promising. Its scope is rather different from that of the hand dynamometer because the elements of intention and visual control are included in the test. It should be quite easy to design pursuit meters with controls suitable for the spastic child and with a display system which they could comprehend easily.

Meanwhile dynamometer records have been obtained in spastic children which display some features of the motor disorders encountered. (*Examples were shown*).

For example, in a record from an 8-year-old girl with very mild athetosis, the defect in the capacity to build up tension steadily and the failure to maintain smoothness of repeated efforts is shown.

Similar defects are seen in some children with cerebral palsies without athetosis. The capacity to synchronize the movements of the two hands is also affected in these children. A most interesting phenomena is seen in two recordings, both taken from 15-year-old children with affection of all four limbs.

The first of these showed that repeated

movement is more consistent in power and timing in one hand when it is used alone. The performance is better than when both are used together. There is incomplete relaxation when both are used although the hand relaxes quite well when it is used alone.

The second of this pair showed a similar defect. For purposes of comparison the last record displayed incomplete relaxation in a condition of different actiology, myotonia dystrophica. The regularity of movement is normal.

The last two records might indicate that there is a difficulty in bilateral control which leads to the preferential and exclusive use of one hand, a phenomenon sometimes seen in cerebral palsy, and in fact this was present in the second subject. It should also be observed that while the strength of the hand grip may be very considerable and even near normal there is intermittency in its production which is destructive to the quality of movement. This may occur in children without much spasticity whose limbs at times are seen to be hypotonic.

Furthermore the question raised by the similarity of some of these records with those of normal children of lesser age is that the nervous system may learn the incorrect way of executing muscular movements just as it learns the correct way in the normal. In other words pathological patterns of movement may be acquired or laid down during the important stages of the development of motor skill and hence be ineradicable later on in life. It may only be possible to prevent the development of pathological movement, never possible to remove it.

" . . . A second difficulty is this. As a student of symptoms you want to question the patient in detail about the character and distribution of pain, its times of occurrence, its relation to various circumstances, when it began and so on. Unless you do this you will get nowhere. But you will find the average patient has very little interest in this procedure. He is indeed apt to be impatient with your enquiries for he is all the time thinking of something else, which to him is far more important. I have a pain, he is saying to himself, and what I am bothering about is not the answers to all these questions but what the doctor is going to do to take my pain away. There is in fact a discord between doctor and patient at this stage of their acquaintance which is inevitable and natural, and must be recognized by both before any more profitable relationship can be established. Sympathy alone can bridge the gap. It is a very apt word in this connection. To understand a patient's symptoms you must literally suffer with him as far as you can. When he tells you that his pain is like toothache you should remember your last toothache; or if he says it is a cutting, or a throbbing, or a bursting or a burning pain, or a pain like a tight band round some part of him, you should draw on your personal experience of such pain to help you to imagine just what he is suffering. Sympathy in its less literal sense—being sorry for the patient—is just as important. You will not obtain an accurate history of symptoms without it, but then if you are not sympathetic in this sense of the word you should not be a doctor at all"—SIR CHARLES SYMONDS. *Canadian Medical Association Journal*, 1951, 65, 422.

4. The Stretch Reflex in Man

A study by electromyography and dynamometry (Strain Gauge). A contribution to classification of the various types of Hypertonus in cerebral palsy.

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Paris.

It is a common opinion that it is not easy to diagnose the various types of hypertonus met in C.P. children. However, it is necessary to define and determine accurately the degree of the hypertonus. Usually it is only noted that the hypertonus is "severe, moderate or mild" without more accurate data. This results in difficulty in appreciating improvement obtained by drugs, rehabilitation or surgery. This was why we have been doing this research, made possible by a grant from the United Cerebral Palsy Research and Educational Foundation.

Hypertonus of cerebral origin may appear under different circumstances:

(1) Some may be seen on passive movement of the limbs of a child lying in the supine position.

(2) Others only appear if more complex postural reflexes come into play, such as the tonic neck reflexes, the sitting position, the standing position or walking.

(3) Others exist only, or at least increase a great deal, during voluntary movement.

(4) Still others appear chiefly under emotional disturbances or sensory stresses such as strong light or loud noises.

Methods

The child is placed in a supine position. We try to avoid interference of both symmetrical and asymmetrical tonic neck reflexes by keeping the head in a central position without extension, flexion or rotation. The child had been gently accustomed to the situation in order to remove all emotional disturbances.

A stretch reflex of forearm flexors is provoked by passive extension of the elbow, done at various speeds.

The angular velocity of extension is calculated from the output of a potentiometer connected to the arm. Two bars fixed one to the arm, the other one to the forearm with their axis the same as the axis of the elbow. A rheostat is connected with this axis, and the variations of electric resistance are amplified and recorded by one of the pens of an electromyograph apparatus. Since the paper runs in the apparatus at speeds of 25, 50, 100 and 200 mm/sec., it is easy to calculate the angular speed.

The stretch reflex is recorded by three ways: High speed camera, electromyography and strain gauge.

1. High-speed camera recording 500 to 1000 frames a second. This research is chiefly interesting in judging diminished stretch reflexes as in ataxia.
2. Electromyographic records were registered by external electrodes. Concentric needles are not suitable for our research* They are rather painful and may by emotional disturbances provoke hypertonus which may interfere with the stretch reflex. They are impracticable during fast passive movements, being able to produce muscle injuries. And, third, the record of motor units are of no interest for us because we have to know as far as possible the function of the whole muscle during the stretching.
   In order to avoid as far as possible superficial skin electrical potentials, (artefacts), we never record if the resistance is greater than 10 Kilohms.
3. The strain gauge evaluates the strength of the stretch reflex. This appliance is made of a blade of copper, the resistance of which changes if it is lengthened. The variations in resistance are amplified and

*These objections to concentric electrodes would not be accepted by all workers.—Ed.

recorded by one of the pens and the strength of muscular resistance is recorded at any moment and without delay. The record is an oscillatory one of 50 cycles, the amplitude of which is linearly proportional to the force.

RESULTS

I. ELECTROMYOGRAPHY

E.M.G. Data in Normal Man.—It is not always easy to obtain complete muscular relaxation in a normal child and even in a normal adult. This total relaxation must be established before any stretching is done. When we speak of complete relaxation we understand a flat record, that is to say, lower than 10 microvolts. It is more difficult to obtain flat record during stretching. Nevertheless, a well trained practitioner always achieves this if the angular speed is lower than 30 degrees in 100 millisec. A flat record is not produced by fatigue of the muscle, since potentials vary from one stretching to the next. We have examined the reaction of the biceps in 65 normal adults and children over five. The flat record was always obtained for both low speeds (about 30 degrees in one second) and high speeds (lower than 30 degrees in 100 millisec.) The necessary number of stretchings to get a flat record was generally 10 or 20. Statistical study showed that it is abnormal to obtain no flat record in 60 testings at a level of $P = \cdot 05$.

E.M.G. Data in Spasticity.—In spasticity it is never possible in 60 testings to obtain a flat record if the passive motion is fast. It is always possible in 60 testings to get a flat record if passive motion is slow, and at rest also. Records registered during the fast stretching are remarkably similar from one stretching to another. It is possible to find the *limit speed*, over which it is impossible to obtain flat records. Under this limit, just as in normal status, less than 60 testings are needed to obtain the flat record. *This limit speed defines spasticity.* In one definite case, two different practitioners have found the same figures after a delay of 2 months. The limit speed may be lowered by drugs.

E.M.G. in Rigidity.—The findings are the same as in spasticity, if there is no additional tremor. Before any stretching it is possible to get a flat record; it is also possible to obtain it during stretching, but it is very difficult. The limit speed is very low, for instance in one case one must move the elbow 30 degrees in not less than 26 seconds to get a flat record.

E.M.G. Findings in "Tension", The hypertonus of athetoid C.P.—By tension we mean the hypertonus seen at times in cases of athetoid cerebral palsy. Strictly speaking, these are of two sorts. There is hypertonus which occurs during the involuntary active moments—during the athetosis. There are athetotic hypertonuses. There are also in the same cases hypertonuses which occur in the absence of any involuntary motion. These might be called non-athetotic athetoid hypertonus. (*Athetosis* indicates a specific type of involuntary movement; *athetoid* is the adjective applied to those cases of cerebral palsy in which athetosis occurs.) Clinically these hypertonuses are very variable; at one moment they are of the utmost violence, a second later weak or absent. Paradoxically, this form of hypertonus often disappears during a fast stretching. With the E.M.G. these clinical data become obvious. There are often high potentials before any stretching. During stretching, the record is very irregular and a flat record may be obtained during the fastest mobilization.

II. DYNAMOMETRY BY STRAIN GAUGE

Normal Status.—We must first make clear that the force recorded by a strain gauge during passive motion is not only due to active contraction of the muscle stretched. During the period of acceleration, force is necessary according to a well-known mechanical law. During the period of constant speed, force due to fric-

tions may intervene. As a matter of fact, in normal persons, the force recorded decreases during passive motion.

Rigidity.—From the beginning to the end of the passive movement, a force is recorded; it falls abruptly to zero when the passive movement has a brief step back; it becomes again a little positive for a brief while and then falls permanently to zero. We used to record 50 to 60 successive stretchings and to have 5 or 6 series in total an average of 300 measurements. We plot for each experiment the angular velocity and the force at a point preceding the end of the passive motion. Speeds are in abscissa, forces in ordinates. In order to have an easier calculation we write in fact not the figures of forces and speeds, but the logarithms of those figures. Two facts ascertained are: (1) The points are sharply grouped; they are distributed along a straight line. It had been possible to calculate a formula so that $F=KV^n$ F being the force, V the speed. Parameter K and exponent ^{n} define the degree of rigidity. The correlation of the formula is very high; $r=0{\cdot}90$.

Spasticity.—In spasticity, the first part of the record is nearly the same as in rigidity, but after a delay the force increases and to a higher degree than in rigidity. The most important fact is that the decreasing of the force is not abrupt but slow when the passive movement is finished, it is to say when the stretching is only maintained, and no longer being increased. If we plot the logarithms of speed against the logarithms of the force, we have again a straight line with a sharp grouping, with the same formula $F=KV^n$ and $r=0{\cdot}89$. The decrease in the force after the end of passive movement has to be calculated because it is the most representative figure of spasticity. In some cases the decrease is rather fast, and in other cases very slow.

Tensions". (The hypertonus of athetoid C.P.)—The variability, clinically observed becomes very clear: for the same speed, the forces recorded are in all cases dispersed, in some cases highly so. The points plotted with the coordinates Force/Speed are more or less scattered. In order to have representative picture of the degree of tension, we have now to calculate the scattering. We hope soon to reach this aim with spatial representations with 3 coordinates. It is already possible to ascertain two facts: (1) It is easy to distinguish the hypertonus of athetoid cerebral palsy from both spasticity and rigidity in the typical cases, but there are intermediate forms; (2) the scattering is obvious on the log paper and eventual improvement by therapy may be measured.

Conclusion

We have now objective methods for distinguishing the different types of hypertonus of cerebral origin and for measuring their degree and consequently the results of therapy. Nevertheless, up to date measurements have been made only on the flexors and sometimes extensors of the forearm. Other muscles should be tested.

Chiefly we have tested muscles in *one* condition—that is, passive movement in a supine patient. It would be necessary to test muscles during postural reflexes (for instance, tonic neck reflex), during voluntary movement, and during other conditions.

The first approach in this way suggests that it is sound to contrast the predictability of both spasticity and rigidity with the capriciousness of the "tension" hypertonus in athetoid cerebral palsy.

SUMMARY

The classification and assessment of the various stiffnesses met in C.P. children are not accurate enough. This is why the myotatic reflex has been studied during passive mobilisation. The E.M.G. study shows that in normal it is always possible to obtain under this condition a flat record if the velocity of the stretching

does not exceed 30° in 100 ms. In spasticity it is always possible to obtain such an absence of potentials at rest, and too if the passive movement is slow enough. There is a definite limit speed which defines the degree of spasticity. In rigidity this limit speed is very low. In athetoïd tensions it is impossible to define any limit speed. Potentials vary from one moment to another.

Dynamometric study with strain gauge brings out the specific pictures of rigidity and spasticity. The force is a definite function of the speed in both spasticity and rigidity. In athetoïd tensions, on the other hand, capriciousness is characteristic, giving an extreme scatter when force is plotted against speed.

High-speed camera records also give interesting data in the measurement of the myotatic reflex in man and hence help to differentiate different types of stiffness and measure them.

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" . . . the exploration of the child's potentialities, rather than the exploration of his weaknesses, is the primary clinical and educational task . . . As Thorndike said long ago, the chief fact about classification of people on the basis of ability is the discovery of waste, that is vast untapped potentials. I am not referring to the squeezing of most of the juice out of the child in terms of achievement, but rather to the fullest enhancement of his life . . . A built-in sense of adequacy and fulfilment is only possible on a realistic basis, that is, by enabling people to do actually what they really can do . . . a healthy personality is largely an expression of a time and place, a system of interpersonal relations involving acceptance, affection, encouragement and belief in one another".—GORDON MURPHY. "Self Realization and Mental Health." *Bulletin of the Menninger Clinic*, May 1959, 23, 81.

" . . . Likewise, all studious, good and honest men, do never suffer their mind so to be o'rwhelmed with the passions of indignation and envy, but that they will patiently hear what shall be spoken in behalf of the truth or understand any thing which is truly demonstrated to them; nor do they think it base to change their opinion, if truth and open demonstrations so persuade them, and not think it shameful to desert their errors, though they be never so antient, seeing they very well know that all men may erre, and many things are found out by chance, which any one may learn of another, an old man of a child, or an understanding man of a fool . . . "—WILLIAM HARVEY. *De Motu Cordis*, 1628.

REHABILITATION CENTRES

Rehabilitation centres were first introduced in this country in 1941 for the training and resettlement of the disabled. Their aim was to help the war effort by bringing every possible man and woman into employment. Since then a number of Acts of Parliament have come into being designed to strengthen the principle of finding work for disabled persons, generally by extending existing acts or including provisions in new acts for the benefit of the whole population. Thus the work done during the second world war has not stopped, as happened after World War I.

It has not been easy to find out how many disabled people would benefit from a rehabilitation course. The British Medical Association's estimate was 50,000 a year, but this figure might be considerably higher if general practitioners knew more about the facilities available, and if regional medical officers generally set out the details of these facilities in their reports.

Courses are provided by 15 Ministry of Labour rehabilitation units, only one of which is residential. One new centre is being prepared. None of these centres is in new buildings. The capacity of these units is 1,600, and 10,000 people a year pass through them directly into industry or a training centre. They perform a most useful function and are generally adequate in number, though the Piercy Committee recommended that some more should be supplied for the important industrial areas that are still without them. It also commented that doctors should not wait until their patients were fully fit for work before allowing them to enter these units. Disabled people are also rehabilitated in hospitals, though often in a rather uncoordinated fashion, more because of the patient's need of therapeutic diversion than as part of any planned rehabilitation. There is a further need in rural areas for rehabilitation clinics associated with a hospital.

The most interesting of the Piercy Committee's recommendations was for building two or three experimental comprehensive rehabilitation and assessment centres, to overcome the confusion resulting from the two different approaches to this problem. One of these centres is now being planned by the Ministry of Labour and the hospital board of the Belvedere Hospital in Glasgow. For people outside the employment field, who are provided for by the local authorities alone, the committee recommended that social centres should be developed and hostels built for the temporarily and permanently disabled. A new American book, "*Rehabilitation Center Planning—An Architectural Guide*", by Salmon & Salmon (Pennsylvania State University Press, $12.50) will interest anyone concerned with the construction of buildings of this kind. It is written for architects by two architects, and is intended to help lay down principles of design for rehabilitation centres as built in the U.S.A. These centres seem much more comprehensive than any in Britain and it will be interesting to see whether the plan of the new Scottish experimental centre will be on these lines.

The authors start by surveying the various factors in the local community which influence the construction of a building. The emphasis is certainly on "local" from both a financial and an ad-

ministrative point of view; half the operating income comes from fees paid by the patients, supplemented by the sale of goods made in the centre and by voluntary effort. Planning principles are discussed in detail, together with a diagrammatic layout of a whole centre whose various elements are later examined most thoroughly. These elements are tabled so that it is easy to see how much noise is permissible, the relationship of various entrances, the preferred exposure, the degree of isolation and the relation to outdoor activity. A further table, setting out the recommended illumination intensities, will come as a surprise to those who are used to the twilight considered adequate in this country.

Then there is a most useful section for anyone planning for wheel-chair patients. This starts with all the essential dimensions of a wheel-chair, followed by diagrams of possible wheel-chair movements, reaching distances vertically and horizontally from a wheel-chair, and the relationship to normal furniture and ramp slopes. The section ends with an examination of door and handle details most sutable for disabled and wheel-chair borne people, window types, sill heights, various wall projections (presumably to be found in existing buildings) and methods of eliminating them.

The authors then mount a hobby-horse called "modular flexibility", to persuade the reader that only modular building materials are really any good for rehabilitation centres, and that repetitive structural units produce maximum flexibility. There is an exploded sectional view of a building showing that a great deal of flexibility is required of the various services, and the point is well made, here and elsewhere, that the number and incidence of disabilities is continually changing, and that only a building equipped with easily moved walls can adapt itself to the varying requirements.

There follow a number of drawings of what, in the authors' opinion, constitutes an efficient and attractive looking centre. Site considerations are next examined, together with the centre's relationship to the community and its lot. Sloping sites are encouraged because easy access can be had to different levels, trees and landscaping are welcomed, but those with built-up city sites must build their centre around inward-looking courtyards, where a pleasant environment can be provided. We are warned to check on zoning, building lines and services before buying the site.

The various elements of the building are now examined in great detail, including all therapeutic equipment, with dimensions and layout. There are a great many extremely useful drawings, and this section will be invaluable to anyone wanting to know, among many other things, the size of a wheel-chair patient scales, a diathermy unit, a portable paraffin bath, a shoulder wheel and ladder, a treatment tank, whirlpool equipment and a hydrotherapy pool. Similar treatment is given to occupational therapy equipment, covering weaving, woodworking, ceramics, sewing, typing, printing and activities of daily living. The section on speech and hearing contains some helpful details of audio-testing rooms. The remaining sections deal as exhaustively with the appliance shop, psychiatrist's rooms, staff-rooms and social rooms. Vocational evaluation and training is explained, plans and dimensions of all the equipment being given. The details of nursing units for in-patients are followed by a section on children's teaching and training, with plans and furniture details; there are some thoughts on administration and general areas.

The book ends with a chapter on environmental considerations, listing a number of points liable to be forgotten; there is a quick look at the future, followed by a useful bibliography. "*Rehabilitation Center Planning*" is likely to remain an invaluable reference book for a long time to come.

EDWARD SAMUEL, A.R.I.B.

LATE READING IN CHILDREN

A REVIEW OF ITS ORIGINS, with DISCUSSION OF A CORRECTING DEVICE FOR THE APHASIC TYPE

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Late reading may be due to twisted perception of letters, seeing "b" as "d", etc. Such "strephosymbolia" has been ascribed to the lateral dominance of hands and eyes being mixed and to the child's failure to establish a necessary dominance. Dr. Barger describes tests for lateral dominance and his method of treating delayed reading in which the pupil starts by learning to read well in a mirror.

Failure of children to achieve reading facility has frequently been ascribed to the sole factors of organicity, of poor teaching methods, or to emotional disorders, including schizophrenic personality-disorder tendencies. The factors enumerated omit one important area which it is the jurisdiction of Medicine to explore. This area is that of "aphasia". (*)

There is accumulating evidence that in addition to organic components in aphasia there can also be in children a developmental etiology of origin, a maturational component in recovery, and a *physiological* element in functioning (Barger 1953. American Medical Association 1952).

Pupils with seemingly adequate exposure to reading instruction sometimes do not respond although primary emotional factors, methodology, and true (organic) mental deficiency have been ruled out of the problem. In such hard-core, resistant pupils a neuro-medical person should be consulted to verify any tendency to twisted perception of reading symbols (strephosymbolia). This condition is classified as an aphasic situation (American Medical Association 1952).

The tendency to twist symbols and the presence of mixed lateral dominance (hereinafter related) were described by the late Dr. Samuel Orton (1937). The condition has been found to interfere with the literate functioning not only of normal pupils but also of hard-of-hearing students, spastic children, sight-impaired pupils, and in upper levels of genuinely mentally retarded children (Barger 1958b). Mixed lateral dominance in function is an endogenous factor (Myklebust 1954, Gesell

*The term "aphasia" is used throughout this article in its generic sense to include varying degrees of impairment in literate abilities, separately or in combination—viz., speech, reading, writing, spelling, arithmetic symbolism, consecutive intellectual action, conceptualization, spatial relationships, perception, and interpretation of communication.

and Ames 1947, 1950, cited by Gesell 1954) and when unadjusted is a genetic, biological impediment to learning. Orton (loc. cit.) stated that many acts in life can be performed bilaterally, as mastication, deglutition, respiration, etc., but that a child who does not establish a dominance in function can run into trouble when trying to learn to read because of the resultant strephosymbolia. The condition is more prevalent than has heretofore been believed, occurring in approximately 59% of late reading cases reviewed in more than a thousand schoolchildren (Barger 1958a, Shepherd 1956).

Mixed lateral dominance, which has been further elaborated by the author, involves various combinations of functional dominance: right-eyed preference with left handedness, and vice versa; dominant portions of the lower extremity of one side and opposite dominant use of parts of the upper extremities; and mixtures of these with the dominant eye, etc. (Some tests to elicit these tendencies are enumerated below.)

The teacher and parents are usually the first persons to notice strephosymbolic tendencies in the child. Boys outnumber girls in late reading about 3 or 4 to 1 in most investigations, whereas girls seem to exceed boys in arithmetic disability (Barger 1957, 1958). A genetic sex linkage is suggested. The pupil who confuses directions in reading can show both reversals and inversals of words and letters: reversing *was* for *saw*, *on* for *no*, *b* for *d*, etc.; inverting words and letters, as *if* for *it*, *b* for *p*, etc.; and attempting to transpose letters within words when writing or spelling, as *chiar* for *chair*, *potnet* for *potent*, etc.

Simplified tests for mixed lateral dominance, and the child's personal history, as well as his family history, reinforce the examiner's recognition of the condition in the late-reading pupil (Barger et al 1957). The family history frequently discloses mixed laterality in other members, sometimes to the third generation. The examiner determines the pupil's preferred writing-handedness, noting any history of enforced change. The preferred hand for batting or throwing a ball, or for eating might be the opposite to that for writing. Some pupils cannot identify their right hand from their left. These tests for mixed lateral dominance include noting which thumb is on top of the other when hands are clasped and the fingers are alternately interlaced (Monroe 1935). There is a neuro-anatomical motivation for the thumb-fold (Penfield and Jasper 1954a). The examiner also observes which forearm is on top of the other when the forearms are folded, the preferred thigh used for climbing by knees on a chair, the preferred extremity for kicking and for hopping on one foot, which ankle-fold is on top when the feet are crossed, which thigh is superior when the knees are crossed and in which direction the child prefers to turn from standing position when called on to turn round and face the examiner. Constant turning preference has been noted in lower animals (Whitter 1959). The presence of ambidexterity or ambi-optica is recorded; the latter is quite an obstacle in correcting reading reversals. In general, the lower extremities are not as refined in precision as the upper ones and the eyes. The right and left preferences of the child are not added up to know which exceeds the other; the examiner wishes to determine whether a mixed condition is present. A single deviation—as the thumb-fold, or the ankle-fold—has sometimes been found significant when associated with late readers—the latent tendency to twist symbols is present. Gesell, at Yale University, in a small series was able to predict by tonic neck reflex tests the future lateral preferences of children, recorded through cinematic studies taken at the ages of one, five, and ten years (Gesell 1954). Laterality tests should be performed individually with children, since in a group the children might discern the object of the tests and try to feign responses.

Not everybody with a mixed laterality

of function finds difficulty in learning to read. This is because those people have made adjustment to the condition. It is the late reader who has not been able to adjust internally to his directional confusion who is in trouble when he attempts to read, because he cannot discern the symbols correctly in perceptive cerebral areas. There is evidence in these late readers that the cerebral mechanism which permits proper orientation is not adjusted and performing (MacDonald and Kegan 1928, Starling 1926).

Experienced psychologists, clinically orientated and eclectic, recognize that an intelligence quotient score on a child who manifests some aspect of aphasia is not entirely valid. A more sure and usually higher intelligence rating than that obtained is obscured because certain pathways of communication are closed in the aphasic pupil. In such cases, the clinical evaluation of the pupil's brightness is more valid than a formal, scored result. A scored I.Q. is not a figure which represents *the* intelligence, but is representative of *many* intelligences and abilities. Acceptance of one flat scored figure as *the* intelligence can obscure recognition of component abilities in the aphasic pupil, and in some cases will cast doubt on the nature of his retardation, causing confusion with mental defect.

The potential functioning ability of the child should be sought. Some word lists of grade achievement in reading give a pupil a 1.3 grade level if he recognizes one word. This can be erroneous as a true measure of the pupil's grade achievement since he might accidentally have known just that one word on the list. The functioning level in reading, rather than the scored grade achievement level, is preferred by teachers as more accurate in representing current performance in reading skill. Educable children should not be fettered with the fetish of a figure.

Psychotherapy and customary remedial reading, despite their excellent techniques, do not relieve the intractable late reader with a mixed lateral dominance; with his unresolved strephosymbolia the pupil is unable to orient literate symbols internally.

The Mirroreading Board

The "Mirroreading Board" (Barclay 1959) and the mirror technique were designed to compensate and assist the late reader whose disability is associated with mixed lateral dominance and strephosymbolia. The mirror technique does not supplant other teaching techniques; rather it employs them, but in conjunction with the mirror. The mirror technique is not a do-it-at-home device; an experienced remedial reading teacher is oriented in the use of the technique is necessary for the tutoring. She knows when to discontinue use of the mirror lest there result some confusion of the confusion with symbols. Some children respond quickly in weeks to the technique, others in terms of months. When the mirror has effected an internal adjustment for the pupil to his directional confusion, the mirror is discontinued. Children who present more than one disability in the aphasic area (see footnote, first page of article) usually require longer to surmount their strephosymbolia. It is interesting that all young children, as those in the first grade, are able to read mirrored images of reading material (Barger et al 1957) but lose this power as they effect an orientation for dominance internally. The child who does not establish such an adjustment to a mixed laterality evidences an impediment to reading by his strephosymbolia.

The mirror employed to correct the deficit in perception is set in a mirroreading board (see fig.) The child peers over a barrier which conceals the reading material as it lies on the board, looking at the inverted image of the reading material in the mirror at the other end of the board.

The principle, in brief, is that the mirror serves as a set of external glasses; it inverts and reverses individual letters of the printed material so that the late reader

who cannot orient the material properly internally is able to view the mirrored image of it as he needs to comprehend and retain it. The inversion of the mirrored image permits the direction of the entire sentence to proceed in the customary left to right direction. Cerebral repositories for reading ability have generally been assigned in Brodman's area No. 39 near the angular gyrus of the dominant hemisphere of the human brain (Barger *et al* 1957). [The cerebral topography compiled by von Economo is held to be even somewhat more accurate in its parcellation by some investigators (Penfield and Jasper 1954b).] Associated with the literate area is one for body orientation, up-down and right-left†.

The mirror technique is for the use of the individual child who requires it. Many strephosymbolic children who had received two years of excellent psychotherapy and remedial reading tutoring were still unable to read. Later when the same teacher, same material, same methods, and same child utilized the mirror technique, these hard-core, reading-resistant children responded. They, themselves, thereby served as more complete controls for the efficacy of the technique than mass comparisons with unrelated "control groups" would have done. As a rule, pupils below the secondary-school level respond more easily to the mirror than do other children. In older students, the accrued secondary emotional frustrations tend to have become fixed and to interfere with flexibility of response to the technique.

About 400-500 pupils have now been assisted in reading with the mirror technique. The necessity for neuro-medical supervision is high-lighted when it is recalled that the subject deals with the ramifications of aphasia in its generic sense. A partial conceptual aphasia, which I believe is related to a special spelling disability, can particularly thwart progress in the late reader. Nevertheless, good readers can remain poor spellers.

Mirroreading board in use

A word should be said about the spatial perceptive powers of children with adequate intelligence who have Little's disease, or other forms of cerebro-spastic conditions. When reading, the nuances of spatial discernment in these spastic children can be rather easily interfered with. The difficulty does not seem to be the impingement of retinal imagery, but rather an aberration of internal perception at the cerebral level. With some of them, a slight tipping of the textbook when held by unsteady spastic hands can cause the cross-bar, for example, of the letter "e" to disappear, and the result resembles an

†See Anotomy Charts published by Charles E. Thomas.

Random Examples Selected From 103 Cases (Boys: 3. Girls: 1.)

Case No.	I.Q.	Grade	Age (years)	Reading Grade: Standard Test.		Functioning Level in Grade		Duration of Daily Instruction With Mirror* (months)	Personality Adjustment
				Original	Final	Original	Final		
Boys:									
1	106	2	8	Pre-primer	1·5	Pre-primer	1·5	1	Less inhibited; less timid
2	87	5	11	Reading readiness	2·2	No basic vocabulary	2·0	9	Can now concentrate and attend; now not fidgety
3	92	3	8	1·3	4·6	Primer	4·0	4	Now happy, well adjusted
4	111	6	12	3·5	6·0	3·0	6·5	7	Reading success restored personality status
5	110	3	8	1·6	4·7	Primer	5·0	3	Now feels secure in reading accomplishment
6	85	6	12	2·8	4·0	2·0	4·0	1	Changed from belligerent, unsocial, unhappy child to friendly, co-operative one
7	88	6	12	2·0	4·0	1·5	4·5	8	Can now accept criticism
8	109	2	7	Reading readiness	2·6	No basic vocabulary	2·6	4	Now not inadequate; contributes to plans
Girls:									
9	112	4	9	2·0	4·7	1·0	4·0	2	Remarkable change to affectionate girl
10	87	4	9	1·9	3·1	Pre-primer	3·0	8	Interest aroused in other school subjects
11	138	2	7¼	1·0	3·1	1·2	2·8	2	Introvert conduct ceased

*Dr. Louise T. Ryan, assistant superintendent; Francis A. Carroll, principal, Public School 147 Queens. Alice B. Speer, principal, Public School 140 Queens. Reading teachers: Sylvia Teschner and Rose Hirschhorn. Intelligence quotients by New York City Bureau of Child Guidance, Ruth I. Lavin, reading supervisor.

"o". Parts of a line of type can disappear from the printed page when the proper spatial view is accidentally altered (Reisenweber 1959). An elaborately equipped room, complete with rheostat for gradual lighting intensities, and with desks and chairs hydraulically controlled for proper elevation of pupil and textbooks are used to compensate for the spastic child's inability to hold his reading material appropriately (loc. cit.).

Depth perception also seems to play a part with the mirror technique. From physics we know that an object placed in front of a mirror appears in image as far behind the glass as the actual object is placed in front of the mirror—that is, the apparent distance from the viewer's eye is doubled. In earlier experiments with the mirror technique it was hoped to give instruction to a group of late readers assembled at one sitting by employing a stereoptician projector. The reading material was appropriately projected on a flat screen. The spatial element of depth provided by the mirror was absent, and the pupils said they preferred their individual mirror alone with its double-depth effect. A quality of spatial depth perception seems to be involved.‡

The limits of this paper allow me to say only what the mirror technique essentially is, rather than to explain how instruction is conducted with it. The "How" is a separate dissertation, and involves the methodology of remedial reading techniques performed by teachers oriented in the mirror technique as well as in phonetics, phonics, word-attack, word-families, word-drill, etc.

The table shows some of the results obtained with children who were late readers and who were tutored with the mirror technique. It will be noted how secondary clusters of emotional manifestations dissipate when the primary core of the reading disability is cleared. When the children have adjusted to the mixed lateral dominance this has become an internal physiological phenomenon; external laterality usage remains as it was previously, but an internal adjustment to it has occurred. It is this internal adjustment for the pupil which the teacher strives for, and which the mirror provides.

‡Junior High School 47, Manhattan. (School for the deaf). N.Y. City Board of Education. Harriet F. McLaughlin, principal; Margaret Wallin, Assistant principal and supervisor of experimental classes for children with language disorders (aphasic children).

SUMMARY

Late reading-ability can be assigned to several origins, including organic disorders, emotional blocking, personality factors, and faulty teaching techniques. In addition, the area of aphasia (in its generic sense and not necessarily of brain-damage origin) can be responsible, and this area should not be overlooked in the diagnostic evaluation.

The mirror technique described here is employed with late readers when strephosymbolia (twisting of symbols) and mixed lateral dominance are present in persistent late reading ability. The technique produces the best results when there are no collateral aphasic handicaps with the reading disability, particularly conceptual aphasic elements.

Some children whose spelling is poor can yet become good readers.

Late reading is a subject with which neuro-medical personnel should take a wider interest. The guidance of such personnel is important in assisting reading teachers in the use of the mirror technique.

The technique has assisted late readers among handicapped pupils as well as normal ones. Its use with cerebral palsied children who are late readers has been limited. More extensive exploration with the technique is desirable among spastic pupils who present directional confusions.

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" . . . In all fields of life, all people, whatever their creed or race, admit what I call the philosophy of the first-rate. I have already quoted John Morley's saying that an educated man knew when a thing was proved and that an uneducated man did not know. I would prefer to say that an educated man knows, and an uneducated man does not know, what is first-rate, and that the best educated man is he who knows the first-rate in the most important human activities. . . . How do you recognise it in practice? There is only one way of getting to know it—by seeing it; and that is the way in which it can be taught. I imagine that the medical student will learn more by seeing a great surgeon at work or by going round the wards with a great doctor than all the text-books in the world can teach him. It is the same in anything. People learn what is first-rate by contact with it. You learn what is great music by hearing it, what is great architecture by seeing it. You learn first-rate business methods by working in a first-rate business firm. You acquire a good taste in wine by drinking good wine, in good cigars by smoking them.

In cigars and wine, if we have the money, in art and music, even if we have not, it is easy to come across the first-rate. But where do we find it in character and life? It is at hand; in the great religions and the great moral systems, but also in literature and history, though here the pure gold runs in veins amid much worthless matter. . . . You may ask, how do you know what is first-rate in literature and art, and still more in human character? . . . We are safe in accepting the judgment of time; it is rarely reversed on appeal."—SIR RICHARD LIVINGSTONE. "What is Education?" *British Medical Journal*, 1953, ii, 456.

EFFECTS OF ABNORMAL BRAIN DEVELOPMENT ON FUNCTION

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There is evidence that many cases of simple spastic diplegia originate in a developmental error. This may not arise during pregnancy but may be specifically brought about in the neonatal period in an incompletely mature nervous system.

When interest was first focused on "cerebral palsy", about 100 years ago, it was thought that abnormalities of parturition accounted for nearly all the cases. Many people still hold this view, and it is an article of faith difficult to dispel. "Cerebral palsy" may be defined as a motor disorder due to non-progressive damage to the encephalon, occurring before the growth of the nervous system is complete. Consequently this disorder is linked with brain development and maturation. We recognise that in a number of patients—for instance, in the 10% of cases acquired after birth—the cause is relatively indiscriminate in its action. Therefore, while it is true to say that there are developmental repercussions of such an indiscriminate damage, the damage itself is not strictly geared to any specific developmental mechanism. This same consideration applies to some causes, such as mechanical trauma or some instances of hypoxia, which act, shortly before, during, or shortly after birth, and to some cases which owe their origin to hypoxic or other indiscriminate insults in utero, well before the time of birth.

But there are a proportion of cases of "cerebral palsy" —possibly a third or so —which are presumably developmental* in origin. These cases presumably arise from causes that interfere with or produce a deviation from a *specific* stage of normal development. Therefore while in all cases of "cerebral palsy" the resulting cerebral damage has developmental* implications, in these cases the damage is more clearly linked with developmental processes, because a discrete part of development is affected.

Of the half a dozen or so neurological pictures of "cerebral palsy", the condition of cerebral spastic paraplegia/diplegia appears to offer the best evidence of being linked with a developmental aberration. Cerebral spastic paraplegia is a condition of lower-limb spastic paralysis of cerebral origin, without notable functional involvement of the upper limbs. In spastic diplegia patients have spastic paralysis of all four limbs, which, however, is less marked in the arms than in the legs (Little Club Memorandum, 1959). It is usually thought in neurological circles that the difference between the two conditions is essentially one of degree. Certainly this appears to be so neurologically, but the aetiological origins of the two syndromes are apparently somewhat different and on these grounds a separation between them

*The term developmental as commonly used is difficult to define. Clearly anything that happens to an organ or tissue that is still developing in an embryo or foetus or even a child is bound to affect development. To some, however, the term implies events in organogenesis, or even more strictly arrest of organogenesis. I will use the term developmental in a narrow meaning to imply block or deviation of a normal developmental pathway, however produced, but mediated through a specific chemical or enzymic process.

seems justified. Clinically, patients with diplegia more often than those with paraplegia have other neurological complications, such as intellectual impairment, fits, and at times other motor signs, such as involuntary movements.

Some support for a developmental origin of spastic diplegia comes from a study of the inherited forms of "cerebral palsy". Simple mendelian inheritance of "cerebral palsy" is rare. But when this happens the condition is almost invariably spastic diplegia with intellectual impairment, usually marked. This was so in the patients described by Penrose (1954), Hanhart (1936) and Wolfslast (1943), as well as those found by Böök (1956) in a genetic isolate in northern Sweden. It was also so in the patients with ichthyosis, described by Pisani and Cacchione (1934) and better identified by Sjörgen and Larsson (1957) and Richards and collaborators (1957). In these patients we are clearly dealing with an inborn developmental error producing spastic diplegia.

Evidence of a possible developmental origin of cerebral spastic paraplegia comes, indirectly, from its clinical features: the distribution of the paresis, and the absence of fits or severe intellectual impairment. All these characteristics militate against the idea of an indiscriminate noxious attack on the brain. But there is an additional point which suggests a developmental origin for cerebral spastic paraplegia—namely, its association with prematurity. "Prematurity" is internationally defined on the basis of birthweight, the term "premature" being applied to babies weighing 5½ lb. (2500 g.) or less at birth. Most babies in this birthweight group are truly prematurely born, in the gestational sense of the word. The maternal and foetal causes of prematurity, which must have an aetiological significance for the production of cerebral palsy, are many, but so far as present evidence goes, cerebral spastic paraplegia seems to be more strictly correlated with the *degree* of prematurity than with its *causes*.

Taking spastic paraplegia and diplegia together, several interesting facts can be observed. The first is that in the general population the incidence of these conditions rises as the birth-weight falls, so that babies whose birth-weight is 4 lb. or less have a 1 in 20 chance of having cerebral spastic paraplegia or diplegia. The second interesting point is that the curve representing the birth-weight of sufferers from these conditions is bimodal. It has one peak at a birth-weight of about 4 lb. and another at a birth weight of 7 lb. (Childs and Evans 1954, Polani 1958). This bimodality suggests that the diplegics/paraplegics are a mixed group. The heterogeneity is probably aetiological, at least in respect to factors involving growth and therefore weight at birth. Taking each clinical condition separately, it is found that the birth-weights of cerebral spastic paraplegic patients are not distributed bimodally; there is a single peak and the average birth-weight of these patients is 4½ lb. (Childs and Evans 1954, Polani 1958). But the distribution of the birth-weights of the diplegic patients remains still significantly bimodal (Childs and Evans 1954, Polani 1958).

Proceeding to another type of clinical dichotomy, in an attempt to obtain homogeneous groups, and following Churchill's (1958) example, the total group of paraplegic/diplegic patients can be separated into two subgroups. In the "simple diplegia" subgroup, the children are normal or almost normal in intelligence, do not have fits, and have an uncomplicated spasticity involving mainly but not exclusively the lower limbs. The "complicated diplegia" subgroup comprises patients who also have mental deficiency and/or epilepsy and/or additional neurological features like athetosis, etc. Now, as Churchill (1958) also found, in the birthweight distributions of each of these groups all traces of bimodality have disappeared. The patients with "simple diplegia", most of whom have simple paraplegia, have an average birth-weight

of 4 lb. (Churchill 1958, Polani 1958), while those with "complicated diplegia", most of whom have spastic diplegia, have an average birth-weight of 6¼ lb. (Churchill 1958, Polani 1958). Further evidence of the relative homogeneity of the "simple diplegia" subgroup is given by the smallness of the variance of the birth-weights in this group (S.D. 1.28; var. 1.64), which is not much greater than the variance of the birth-weights of a group of normal survivors (Karn and Penrose 1951) and is very much smaller than that of the "complicated diplegia" subgroup.

The meaning of the association between ponderal "prematurity" and cerebral spastic paraplegia/diplegia is difficult to interpret. It is known that most abnormal conditions of the foetus reflect themselves in a decreased birth-weight—though not all babies who are underweight at birth are abnormal (Penrose 1954). It is interesting that radiation injury to the foetus, both in man and in animal experiments, also expresses itself in a general disturbance of foetal growth and a low birth-weight (Penrose 1957). However, a few abnormal conditions are associated with a higher than average or at least fully average birth-weight, notably diabetes and pre-diabetes of the mother (Jackson 1955) and also of the father (Jackson 1954), endemic cretinism (Child and Gardner 1954), and persistent ductus arteriosus (Polani and Campbell 1959). This excessive birth-weight need not always be the result of an excessive foetal growth but can result from pathological conditions causing retention of fluid in the foetus. (For the condition in animals see Hadorn 1955).

In the present state of our knowledge we can do no more than speculate on what may be the possible causes of the cerebral spastic paraplegia/diplegia syndrome. We lack, for instance, information from necropsy studies of brains of children with this condition, particularly of cases of spastic paraplegia or of intelligent diplegics. Birth-trauma as the responsible cause has had many supporters in the past. Present evidence (Eastman and De Leon 1955) tends to minimise its importance as a causative factor in "cerebral palsy" generally and particularly in the group of conditions here considered. Anoxia, usually at birth but sometimes intrauterine, is at present a favourite cause. But the evidence on anoxia is, to say the least, confusing (Tardieu *et al* 1953). Undoubtedly anoxia plays a part in the origin of certain types of "cerebral palsy", for instance, bilateral athetosis not associated with severe neonatal jaundice, or a number of instances of "complicated diplegia". But only rarely can it be incriminated in the causation of "simple diplegia", and only exceptionally in that of "simple paraplegia". In fact in this last condition the only abnormality observed is simple uncomplicated prematurity. So, again, it seems that there may well be a developmental explanation.

The studies of Flexner and his associates (Flexner 1955, Flexner *et al* 1941, 1950) on the "critical period" of cortical development of the guineapig *in utero* have demonstrated the close link between morphological, electrophysiological and enzymic maturation. And Hamburgh and Flexner (1957), in their studies on the rat, have shown how different species vary in the timing of the "critical period". Himwich and his collaborators (1941) have shown that anaerobic metabolism develops according to a definite sequence within the neuraxis, centre by centre. Ashby and her associates (Ashby 1944, Ashby and Butler 1948, and Ashby and Schuster 1950), using carbonic anhydrase as an indicator of relative nervous system maturation, have shown in a similar way the ascending increase of this enzyme occurring in parallel with functional maturation both in animals and in man.

In the light of the work of Flexner and others (1950) it is perhaps significant that electrical activity of the human brain begins at about the seventh foetal month (Tizard 1958, Minkowski 1959, McIlwain 1955). It may be reasonably expected that,

parallel with this, morphological and enzymic changes occur as well. It is probably significant that so many babies with simple diplegia are born at this very time of electrophysiological maturation. And is the act of birth in some way causatively connected with their "cerebral palsy"? Is it not possible that they are not born prematurely because of something causing both prematurity and cerebral palsy (Collier, 1924) but rather that being born prematurely gives some of them cerebral palsy? And is it not possible that this happens only to some prematures by reason of individual peculiarities of biological maturation, so that it is the biological age that matters rather than an arbitrary and too rigid gestation age? One might wonder whether, in this particular group of babies, some part of the nervous system is not in a process of crucial maturation and whether for this maturation the intrauterine environment is essential. If this were so, then the cause of their "cerebral palsy" would be developmental and largely postnatal. Given knowledge of the mechanism the palsy could be preventable. Anoxia, possibly subclinical, might be incriminated. But it would not be too unreasonable to think that an excess of oxygen may be at fault. After all, hyperoxia can probably cause hyaline pulmonary membranes (1958) and it can certainly cause retrolental fibroplasia, the latter probably through a combined tissular and vascular mechanism (Ashton *et al* 1957). And even oxygen at atmospheric pressure and concentration may be harmful to some prematures.

Whether or not hyperoxia is responsible for some cases of "simple spastic diplegia", the fact remains that the incidence of spastic diplegia is much greater than expected in prematures suffering from retrolental fibroplasia.

Urgent Needs

These are speculations. There seem to be two urgent needs: first, to learn more about the mechanisms of normal development and differentiation of the mammalian and human nervous system; secondly to study the brains of simple spastic diplegia patients which come to necropsy. Even then, however, we might still be in the dark. A much more quantitative neuropathological approach than is at present generally applied might be necessary to provide the answers, by, say, a study of relative and absolute numbers of neurons in various areas, or their finer connections. The changes may well be minute and quite limited, as I would expect them to be in those many prematures, cause unknown, whose prematurity does not result in overt neurological handicaps but in a definite reduction in intellectual functioning (Douglas 1956).

SUMMARY

Cerebral palsy is rarely inherited, but when it is, it presents with diplegia and oligophrenia. Because of this fact and the clinical picture of simple spastic diplegia and paraplegia, the association between these conditions and the degree of prematurity, their association with retrolental fibroplasia, and supporting experimental evidence, it is suggested that these forms of cerebral palsy sometimes have a developmental origin.

Specifically they may arise from an arrest of development, presumably postnatal, owing to deprivation, through premature birth, of the intrauterine environment which may be essential to final maturation of the nervous system.

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MEDICAL ASPECTS OF A COMPREHENSIVE SURVEY OF CEREBRAL PALSY

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Eastern Scotland, in surveying its cases of "cerebral palsy," provides an example that other areas might well follow. An attempt is made to characterise a special form of bilateral spastic affection, probably developmental in origin.

DURING the years 1955 to 1957, a survey was made of all cases of cerebral palsy up to 21 years of age in an area of Eastern Scotland comprising the city of Dundee and the counties of Angus, Perth and Kinross. The survey was planned to obtain comprehensive information concerning the medical, psychological and social status of a single group, representing a cross-section of cerebral palsy in the community. A full report is in course of preparation but some medical aspects are briefly discussed in this paper.

Terminology and Classification

Cerebral palsy is here defined as a disorder of motor function resulting from a permanent, non-progressive defect or lesion of the immature brain. Disability from spinal or progressive neuromuscular conditions is thus excluded. Since in my view cerebral palsy may arise at any time until the brain reaches maturity, no upper age-limit has been imposed, but in fact only 6 cases developed between 3 and 8 years of age, and none thereafter.

The principal purpose of the medical examination was to establish a precise diagnosis, and cases were therefore classified according to the physical findings at the time of examination, using a scheme of clinical classification similar to that approved by the American Academy for Cerebral Palsy (Minear 1956). In considering aetiology, however, the cases were grouped into syndromes along the lines advocated by Balf and Ingram (1955), although differing in detail.

General Results of Survey

In the region surveyed, which has a population of about 412,000, the number of cases of cerebral palsy confirmed was 240. This represents a prevalence of 2·0 per 1000 under the age of twenty-one years, a result closely comparable with the findings of other European field surveys in which ascertainment has been very thorough, such as the Edinburgh survey (Ingram 1955a) and the Ostergotland County, Sweden, survey (Herlitz and Redin 1955). In our survey there were 111 females (46%) and 129 males (54%), a ratio of 1 female to 1·16 males. This finding was largely accounted for by the considerable excess of males in spastic hemiplegia (36F:53M) and in athetoid cerebral palsy (6F:12M).

The patients were graded as mildly, moderately or severely handicapped according to their total physical disability, regardless of the type of cerebral palsy.

Such grading must be rather empirical and no criteria were laid down, but, since the assessments were all made by one observer, the standards probably remained reasonably constant. This grading differs from the four categories of the American classification and from the three categories used by Ingram (1955a), who employed different criteria for each form of cerebral palsy. Thus, for example, a moderately handicapped hemiplegic in Ingram's series would be less handicapped than a moderately handicapped diplegic, whereas if I assessed them both as moderately handicapped the disability would be comparable. These differences in method are responsible for the apparent disparity in the results in table I. The high incidence of mild handicap in the Dundee survey is mainly due to the fact that the majority of cases of spastic hemiplegia—the commonest form of cerebral palsy—fell into this category.

Of the 240 cases, 60 (25%) had a history of epileptic seizures, which were more common in cases of postnatal origin (34·5%) than in those of prenatal or intranatal origin (23·2%). Epilepsy, cerebral palsy and mental disorders were not recorded more often in the family histories than would be expected among the general population. There were 2 pairs of siblings in the 240 cases: in only one of the 240 was parental consanguinity established.

The 240 cases were classified as follows:

A. Spasticity: 188 (78·3%)

Monoplegia	12 (5·0%)
Hemiplegia	89 (37·1%)
Double hemiplegia ...	3 (1·3%)
Paraplegia	29 (12·1%)
Triplegia	9 (3·8%)
Tetraplegia	46 (19·2%)

B. Athetosis: 18 (7·5%)

Rotary	10
Choreo-athetoid ...	4
Tension	2
Dystonic	2
Hypotonic	nil
Other types	nil

G. Rigidity: nil
D. Ataxia: 4 (1·7%)
E. Tremor: 1 (0·4%)
F. Hypotonia: 3 (1·3%)
G. Mixed Types: 23 (9·6%)
H. Unclassified: 3 (1·3%)

Thus in general terms four out of five patients had spastic cerebral palsy — spastic hemiplegia in two out of five and spastic tetraplegia in one out of five. These results are in reasonably close agreement with those of most other large field surveys carried out in the United Kingdom. Mixed types of cerebral palsy constituted about a tenth of the whole series. Other types were less common: thus uncomplicated athetoid cerebral palsy accounted for only 7·5% of cases and ataxia for only 1·7%. The three unclassified cases were not seen personally

TABLE I.—Numbers of cases and percentage incidence of mild, moderate and severe grades of cerebral palsy in the Dundee and Edinburgh (Ingram 1955a) surveys.

Grade of handicap	Dundee survey		Edinburgh survey	
	No.	%	No.	%
Mild	97	42·7	52	25·0
Moderate	79	34·8	80	38·0
Severe	51	22·5	76	37·0

and although they certainly had cerebral palsy, and thus had to be included in the total, they were not classified further.

Spastic Monoplegia

Spasticity involving one limb usually represents an incomplete form of hemiplegia or paraplegia. Brachial monoplegia might therefore be classified as hemiplegia and crural monoplegia as paraplegia but for the fact that there may be doubt as to the condition originally present. Thus a patient with a spastic left leg may have had paraplegia with complete restoration of function in the right leg, or such a monoplegia may be the residue of a very mild hemiplegia in which the left arm has recovered more completely than usual. Such considerations make it difficult to be certain of the origin of all monoplegias, and indicate the need for a separate category. Moreover, whatever the original condition of such patients, they present functionally as monoplegics so it seems reasonable to regard them as clinically distinct. There were 2 cases of brachial monoplegia and 10 cases of crural monoplegia in the survey.

Spastic Hemiplegia

There were 89 cases of spastic hemiplegia, 58 (65%) being on the right side. This preponderance of right-sided hemiplegia was found in cases of both postnatal and congenital origin (table II).

TABLE II.—Side affected in various groups of cases of spastic hemiplegia.

Side of hemiplegia	15 postnatal cases	15 prematurely born	21 with abnormal birth at term	34 with normal birth at term	4 with unknown obstetrical history
Right	12	8	13	23	2
Left	3	7	8	11	2

Thus there was no tendency for hemiplegia of intranatal or postnatal origin to be left-sided, as was found by McGovern and Yannet (1947). There was a higher proportion of right-sided hemiplegia in the moderately handicapped group than in the mildly handicapped group, while body measurements showed that growth was more severely affected in the right-sided hemiplegics.

Contrary to the experience of some others, athetoid movements were very rarely encountered in spastic hemiplegia, and the only example in the series was classified with the mixed group.

When the hand was only moderately affected it often retained a good deal of function, but in 22 cases the hand was virtually useless, either because it was small and flabby with weak clumsy fingers (16 cases) or because it was simply not used, even though it seemed capable of considerable function (6 cases). Some of the reluctance to use the hand in hemiplegia is undoubtedly due to sensory disturbances, and of the 54 patients of sufficient age and intelligence to cooperate in sensory testing, 15 (28%) showed some degree of astereognosis, while 4 had diminished sensation of touch and pain over the hand and wrist. Thus the findings confirm that sensory disorders are common in hemiplegia, though the incidence was not so high as that reported by Tizard and others (1954), possibly owing to the greater number of mildly handicapped cases in the Dundee series.

Spastic Paraplegia, Triplegia and Tetraplegia: the Cerebral Diplegia Syndrome

Spastic paraplegia, spastic triplegia, and some cases of spastic tetraplegia are different degrees of a single syndrome — the cerebral diplegia syndrome. Most cases of crural monoplegia represent the mildest degree of this syndrome, but because of the uncertain origin of some

cases, they have not been included. In the tetraplegic cases, the arms are less spastic than the legs, and in lesser degrees of severity the arms may be normal, so that the patient then has paraplegia. In a few cases one arm is normal while the other shows minimal abnormality, and such cases are classified as triplegia. This distinction is important when employment is being considered, for the possession of one functionally normal hand may make all the difference to employability. The number of paraplegics and triplegics varies inversely with the diligence with which abnormalities are sought, and a number of cases previously diagnosed as paraplegia were classed as tetraplegia in this survey because of minimal abnormality of the upper limbs.

Anyone examining a large number of cases of spastic tetraplegia over a short period cannot fail to be impressed by the variety of clinical pictures encountered. Typical cases of double hemiplegia form a distinct group, with the arms more spastic than the legs, dysarthria and dysphagia, poor mental and physical development, and often a family history of mental disorder and epilepsy; the 3 cases in this survey have been classed in a separate group. Most cases of the cerebral diplegia syndrome can be diagnosed comparatively easily, by the greater involvement of the lower limbs and other features. I recognise yet a third syndrome, characterised by very severe mental defect and severe spasticity involving all four limbs to about the same degree. Rather than coin a name, I call this syndrome "Type II spastic tetraplegia," to distinguish it from the severe degree of the cerebral diplegia syndrome, which I call "Type I spastic tetraplegia."

In addition to these recognisable syndromes, cases are encountered which do not readily fall into one or other category. Some of these may represent atypical examples of the main syndromes, while others may conform to syndromes as yet undifferentiated. The importance of recognising and separating these syndromes of spastic tetraplegia becomes apparent when aetiology is considered. The principal characteristic features of Types I and II spastic tetraplegia as encountered in this survey are as follows:

Type I	Type II
1. Pregnancy abnormal	Pregnancy normal
2. Premature or abnormal birth	Normal birth at term
3. "Cerebral" signs such as cyanosis and convulsions in the neonatal period	Lethargic in the neonatal period
4. Condition not suspected by parents until one year old or later	Condition suspected in the first six months
5. Stiff, adducted legs (with awkward gait and tendency to lordosis if walking has been achieved)	Lying in attitude of general flexion (with scoliosis and misshapen chest in older patients)
6. Arms less spastic than legs	Severe spasticity of all limbs to about the same degree
7. Head circumference more than 18 inches	Head circumference 18 inches or less
8. Disability moderate or mild	Disability severe
9. Mentally normal or mental handicap grade I or II.	Mental handicap grade III

In an attempt to classify the cases, these characteristic features have been used as diagnostic criteria, although admittedly this is not entirely satisfactory, since, for example, characteristic number 4 may vary in different communities. Nevertheless, for the purposes of this survey, each of the 46 cases of spastic tetraplegia was classified as either type I or type II if it fulfilled 6 or more of the criteria of the type and not more than 2 of the criteria of the other type. On this basis, 19 (41·3%) were type I and 16 (34·8%) were type II; the remaining 11 did not fulfil the requirements for either type, but 3 were probably type I and 3 probably type II, while 2 were too young for their type to be recognisable. In classifying tetraplegia, I have not found the distinction between symmetrical and asymmetrical cases to be a useful one, as some others have done (McGovern and Yannet 1947).

Rigidity and Type II Spastic Tetraplegia

The term rigidity has been abused in work on cerebral palsy, because it has been used to describe such varied conditions as spastic limbs which are stiff as the result of contractures, a stage in the development of diplegia (Ingram 1955b), the type of cerebral palsy which follows kernicterus (Collis et al. 1956) and the infantile motor behaviour of mentally defective children (Woods 1957). Here, the word is used only for those cases in which the dominant physical sign is rigidity of the classical Parkinsonian type, often described as either "cog-wheel" or "lead-pipe" type. Rigidity in this limited sense was encountered in only one case in the survey, when it was associated with tremor. It is difficult therefore to account for the fact that Phelps, whose use of the word rigidity (1954) appears to fit in with this concept, regards it as "perhaps a fairly common type". Possibly such cases tend to accumulate at specialised centres such as his and thus give an erroneous impression of their frequency. This suggestion is supported by Phelps' remark that "rigidity with tremor is frequently seen"; such a combination of signs could hardly be overlooked, and yet only one case was found in this survey.

The frequency with which rigidity has been recorded in some other series of cases of cerebral palsy is, I believe, due to cases of type II spastic tetraplegia being regarded as cases of rigidity. Woods' comment (1957) on severe cases of "rigidity"—that "they lie in bed rigidly flexed in a foetal position, in contrast to cases of spastic quadriplegia who, when awake, lie in bed fully extended, often with scissors formation of the legs" illustrates the difference between type II and type I spastic tetraplegia as described in this paper. I prefer to regard the motor dysfunction shown by patients with type II tetraplegia as a form of spasticity, even though it may not always appear to be true hypertonic spasticity. Much of the stiffness observed in these patients develops because their profound mental deficiency results in their lying immobile for long periods, and in infancy and early childhood signs of spasticity, such as the stretch reflex and sustained clonus, are much more in evidence.

Type II Spastic Tetraplegia and Mental Defect

Type II tetraplegia is one of the most severe forms of cerebral palsy, with profound mental defect and sometimes congenital malformations. Between this extreme at one end and the mentally defective child who has no motor handicap at the other, there is a wide range of cases and it is very difficult to define the borderline where mental defect alone ends and mental defect plus cerebral palsy begins. In the survey there were 3 cases of spastic tetraplegia which did not conform to either of the main types but which may have represented lesser degrees of the type II tetraplegia syndrome. In all 3 there was slight but definite spasticity of all limbs to the same degree; all 3 had severe mental handicap; all walked without support, with a "shambling" gait, legs together and slightly flexed at all joints, feet in the valgus position heels off the ground, shoulders hunched forwards, and arms either straight and pronated or slightly flexed. All 3 were born spontaneously at term after normal pregnancies; one had hypospadias. These cases therefore had certain characteristics of type II tetraplegia, the main difference being the less severe physical handicap.

There were 36 children who had previously been diagnosed as cerebral palsy but were excluded from the series as being cases of simple mental defect without cerebral palsy. These patients showed many different kinds of abnormal gait, such as shuffling along with all limbs slightly flexed, walking with feet wide apart, and other variations which can only be described as "clumsy" or "awkward". Nevertheless, they could be remarkably agile and spasticity could not

be demonstrated in any of them. A few cases, it is true, had contractures caused by long inactivity which resembled those sometimes found in cerebral palsy, but usually careful observation enabled the distinction to be made. Another misleading feature was the general briskness of the tendon reflexes often encountered in these cases. However, although in a few borderline cases the decision was very difficult, in general these infantile patterns of motor behaviour in mentally defective children were not regarded as cerebral palsy. In addition to motor clumsiness, Norman (1953) described "choreiform" movements in 20% of mental defectives; if the awkward movements of mental defectives were to be regarded as a form of cerebral palsy (Woods 1957) then these 20% who have purposeless movements would also have to be included, which would widen the concept of cerebral palsy so much as to render it valueless.

Athetoid Cerebral Palsy

Five main categories of athetoid cerebral palsy were used in classification:

1. *Rotary*, describing the common type with writhing, often rather slow rotating movements; this term was used in order not to differ unnecessarily from the American classification.
2. *Choreo-athetosis*, describing quicker, more jerky movements, somewhat similar to those found in Sydenham's chorea, but usually more composite and having a writhing element in them.
3. *Tension athetosis*, when the athetoid movements—whether rotary or choreo-athetoid —are suppressed by muscle tension of such a degree that it is the most prominent feature. The word "tension" is used to describe increased tone, possibly partly voluntary, in all muscle groups, and is preferred to "rigidity" because of the restricted interpretation of the latter term.
4. *Dystonic athetosis*, in which hypertonic postural attitudes and dystonic spasms are the outstanding features, the trunk often being more involved than the limbs: this is usually a developmental stage in athetoid cerebral palsy, but may occasionally persist.
5. *Hypotonic athetosis*, seen mainly in infants, who appear generally hypotonic but show minimal involuntary movements.

Of the 240 cases, 28 had athetoid cerebral palsy, but in 10 of these the athetosis was associated with some other form of cerebral palsy and these cases are included in the mixed category. The 18 cases of uncomplicated athetoid cerebral palsy comprised 10 rotary athetoids, 4 choreo-athetoids, 2 tension athetoids and 2 dystonic athetoids.

The majority of athetoids whose disorder is the result of kernicterus can be recognised by the association with high-tone deafness and possibly ocular defects, but not by the type of athetosis, for rotary, choreo-athetoid and dystonic forms were recorded in such cases in the survey.

Ataxic Cerebral Palsy

This term is restricted to ataxia of "cerebellar" type, characterised by signs such as ataxic gait, intention tremor and nystagmus. Only 4 uncomplicated cases were found in the survey, although ataxia complicated by other disorders of motor function occurred in a further 9 cases, mostly ataxic diplegics.

Hypotonic Cerebral Palsy

Three forms of hypotonic cerebral palsy were recognised:

a. *Generalized hypotonia of infants in the early stages of athetoid or spastic cerebral palsy.* This is not a separate category, but if the ultimate type cannot be recognized, temporary classification as "hypotonic" may be necessary.
b. *Generalized hypotonia with increased reflexes, described by Perlstein* (1952) *as atonic spastic diplegia.* The single case in the survey was placed in the mixed group (hypotonia + spasticity).
c. *Generalized hypotonia with normal reflexes, usually associated with mental defect.* There were 3 cases which were classed as hypotonic cerebral palsy.

Mixed Types of Cerebral Palsy

Of the 240 cases, 23 (9·6%) were classified as mixed types of cerebral palsy, a category which includes all cases with two or more distinct and definite disorders of motor function: minimal dysfunction, such as the hypotonia in certain muscle groups or the slight tremor of effort seen in many spastics, would not of course qualify. This system is considered preferable to classifying cases according to

the predominating disorder, and is helpful in assessing suitability for future employment. The 23 cases were as follows:—

Athetosis + spasticity	8 cases
Ataxia + spasticity	7 cases
Athetosis + ataxia	2 cases
Tremor + spasticity	4 cases
Tremor + rigidity	1 case
Hypotonia + spasticity	1 case

Spasticity in 6 of the first group and all of the second group took the form of the cerebral diplegia syndrome, so that these cases may conveniently be described as having "athetoid diplegia" or "ataxic diplegia", provided it is appreciated that "diplegia" refers to "the syndrome of cerebral diplegia" and not to the anatomical distribution of the athetosis or ataxia.

The 6 cases of hydrocephalus in the survey were all in the mixed group, 3 having ataxic diplegia and 3 spastic tetraplegia associated with gross tremor.

Aetiological Factors

In 29 of the 240 cases (12·1%) the cerebral palsy was considered to be of postnatal origin, the principal causes being acute infantile hemiplegia, kernicterus and meningitis. There were thus 211 cases of prenatal or intranatal origin and complete histories were available for all but a few of them, so that the total number of cases in the discussion which follows is about 200, the exact total being slightly different for each factor analysed.

In considering aetiology, the clinical classification used in the survey is unsatisfactory, since knowledge is sought about the causes of syndromes, not of disorders of motor function. No attempt was made to determine the probable cause in each individual case, but the cases were grouped in syndromes as follows:—74 cases of spastic hemiplegia; 56 cases of cerebral diplegia, comprising 28 cases of spastic paraplegia, 9 cases of spastic triplegia, and 19 cases of type I spastic tetraplegia; 16 cases of type II spastic tetraplegia; and 9 cases of athetoid cerebral palsy (this excludes 9 cases of postnatal origin, among them all those due to kernicterus). The 20 cases of mixed cerebral palsy (excluding 3 of postnatal origin) are also considered together, since, though not constituting a syndrome, they do represent an especially severely handicapped group. The numbers in the other syndromes are too few to make discussion of aetiology profitable.

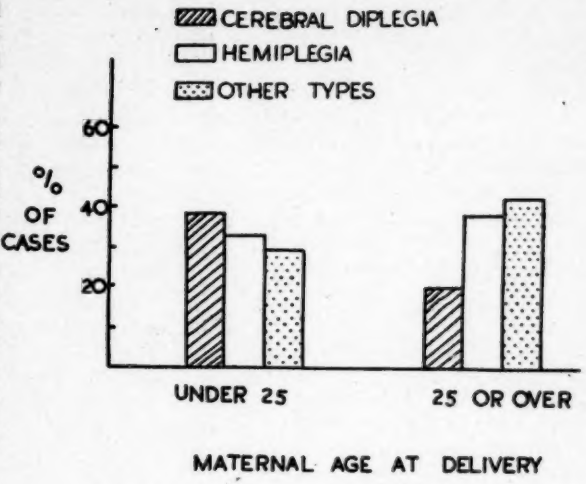
As a group, the mothers of the children with cerebral palsy were older at delivery than the mothers of normal children. Thus, for example, 23·5% of the survey group were 35 years or over when the baby was born, the expected rate being 16·3%. For a number of reasons data from the 1951 Census (Scotland) and the Report of the Registrar General for Scotland (1951) have been used as suitable standards for comparison. Cerebral diplegia was relatively more common in the children of younger mothers, and athetoid and mixed types in the children of older mothers (see figure).

Parity seemed to have little relationship to the type of cerebral palsy, except possibly to athetosis of prenatal or intranatal origin, in which the percentage of mothers of 25 years or over who were primiparous seemed unduly high (55·6% compared with 25—38% for other types of cerebral palsy). The number of cases of athetosis is too small, however, to make this difference statistically significant.

Social class did not appear to play an important part in aetiology. The survey did suggest, however, that it was most unusual for the first child of a young mother in Social Class I or II to have any form of cerebral palsy, and that cerebral palsy—especially cerebral diplegia—was rather more common than expected among mothers in their early twenties in Social Classes IV and V. Neither of these findings was statistically significant at the 5% level.

It is difficult to assess the significance of abnormalities of pregnancy, such as

haemorrhage or maternal disease, particularly in pregnancies which terminated prematurely, in which they were especially common. In cases where the infant was born at term, however, abnormalities in the first three months of pregnancy were nearly three times as common in those in which the birth was normal as in those in which there was an abnormal delivery, and this suggests that such abnormalities may be important in some cases of cerebral palsy. Table III shows that haemorrhage in pregnancy was especially common in cerebral diplegia and in mixed types of cerebral palsy. In cerebral diplegia the haemorrhage occurred in a younger group of mothers (average age 24 years), and 90% of the haemorrhages were in pregnancies which terminated prematurely. In mixed types of cerebral palsy, on the other hand, the average age of the mothers who had haemorrhages was 30 years, and none of their infants was premature. Table III



Effect of maternal age on relative incidence of different types of cerebral palsy in 186 cases, comprising 58 under and 128 over 25 years.

TABLE III.—Incidence of various factors of possible aetiological importance in cerebral palsy.

Factors		Cerebral Diplegia	Spastic Hemiplegia	Type II Spastic Tetraplegia	Mixed Cerebral Palsy	Athetoid Cerebral Palsy
Mothers aged 25 years or more at delivery	No.*	29/52	49/68	11/15	16/20	9/9
	%	**55·8**	**72·1**	**73·3**	**80·0**	**100·0**
Mothers having haemorrhage during pregnancy	No.	11/54	6/69	1/16	4/19	0/8
	%	**20·4**	**8·7**	**6·3**	**21·1**	**0**
Infants weighing 5½ lb. or less at birth	No.	33/54	15/69	1/16	0/19	0/8
	%	**61·1**	**21·7**	**6·3**	**0**	**0**
Forceps (excluding breech) deliveries	No.	12/56	9/70	2/16	5/19	4/8
	%	**21·4**	**12·9**	**12·5**	**26·3**	**50·0**

*The second figures, which indicate the total number of children in each group, vary slightly according to the information available.

also emphasises the well-known association of prematurity with cerebral diplegia.

The incidence of abnormal birth among mature infants was 41·7%, and delivery by forceps was by far the commonest abnormality. Analysis of the data showed that in premature infants the number of forceps deliveries was little different from that expected, whereas in mature infants the rate was much higher than normal even in groups of comparable maternal age and parity. The data indicate that delivery by forceps was important in the aetiology of most forms of cerebral palsy but especially in athetoid and mixed forms (table III). This does not mean that the application of forceps per se is a cause of cerebral palsy, because during the period under consideration forceps were usually applied only in difficult deliveries and the effects of the forceps cannot be distinguished from the effects of anoxia and trauma associated with such deliveries. More frequent delivery by low forceps, as now practised, may in fact prevent damage and so paradoxically reduce the incidence of cerebral palsy.

The comparatively low rate of forceps delivery in spastic hemiplegia is noteworthy, since this condition was formerly often regarded as the prime example of birth injury. In this survey, whereas prematurity, abnormal birth or postnatal disease provide possible explanations for all but 14% of the cases of cerebral diplegia, these factors were absent in no fewer than 38·2% of cases of hemiplegia, a finding similar to that of Brandt and Westergaard-Nielsen (1958).

Conclusions Concerning Aetiology

The contribution that a retrospective survey can make to elucidating the aetiology of cerebral palsy is limited, but the results permit the following general conclusions concerning this series:

1. Postnatal causes accounted for about one-sixth of the cases of hemiplegia, about half the cases of athetoid cerebral palsy, and a very small proportion of all other types. The proportion of cases of athetoid cerebral palsy of postnatal origin should diminish greatly in the future with the disappearance of kernicterus.
2. Birth injury, anoxic or mechanical, to mature infants accounted for about half the cases of athetoid and mixed cerebral palsy, a quarter of the cases of spastic hemiplegia, and a quarter of the cases of cerebral diplegia.
3. Prenatal causes unassociated with prematurity accounted for virtually all the cases of type II spastic tetraplegia, about half the mixed cases, and rather less than half the cases of hemiplegia.
4. Premature birth was important in 3 out of 5 cases of cerebral diplegia and in about one-sixth of the cases of hemiplegia. It was of little importance in other types of cerebral palsy (only one case of athetosis was the result of kernicterus of prematurity).

If prematurity causes cerebral diplegia as a result of birth trauma or of maturation arrest (Polani 1958) then the proportion of cases due to perinatal causes rises to 7 out of 8; if, on the other hand, the prematurity and the cerebral diplegia are results of the same prenatal influence, then prenatal causes are responsible for three-quarters of all cases of cerebral diplegia. The evidence from this survey does not provide an answer to this problem.

SUMMARY

In a survey of cerebral palsy in Eastern Scotland there were 240 cases under the age of 21, a prevalence of 2·0 per 1,000.

Clinical classification and grading by severity of handicap revealed relatively high proportions of mixed types (9·6%) and mildly handicapped cases (42·7%). About four-fifths of the cases (78·3%) had spastic cerebral palsy, about 2 in every 5 (37·1%) had spastic hemiplegia, and about 1 in 5 (19·2%) had spastic tetraplegia. An attempt has been made

to distinguish the cerebral diplegia syndrome from a form of spastic tetraplegia, probably developmental in origin, designated Type II spastic tetraplegia. "Cogwheel" or "lead-pipe" rigidity was the dominant physical sign in only 1 case—the only one where true rigidity was associated with tremor. The stiffness seen in this type is largely a result of the immobility arising from their profound mental defect.

Retrospective analysis of the obstetrical histories confirmed the close association between the cerebral diplegia syndrome and premature birth. Advanced maternal age and forceps delivery seemed important in the aetiology of cerebral palsy, especially of the athetoid types. Except perhaps in athetoid cases, primiparity was not important. Of the 240 cases, 29 (12·1%) were of postnatal origin, the commonest causes being acute infantile hemiplegia, kernicterus or meningitis.

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" . . . it is not a multitude or a great mass of books that makes a ready medical man and supports him in his work and ideas. It is the literary treasures and the valuable current evidences which fit his personal efforts, help develop them, and please him—apart from the factual needs involved in his work—and stimulate that vital energy within him, of which his medical interest forms the outward or social expression. If at the same time a few very select books are drawn into his circle of friendship, their purpose is fulfilled." —J. CHRISTIAN BAY. "Historical Mindedness in Medicine". *Bulletin of the Menninger Clinic*, July, 1959, 23, 121.

THE HABILITATION OF ADULT "SPASTICS"

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Owing to the brain damage which is the basis of cerebral palsy, its treatment has to be on essentially different lines from that of other disabilities. These patients often require expert supervision at all times, not only during physiotherapy. In the habilitation of adults the aim should be to improve their social bearing, give them the largest possible measure of independence, and train them for suitable work.

THE term, "*re*-habilitation," cannot rightly be applied to cases of cerebral palsy due to brain injury before or at birth, because these people have never been habilitated. This is worth emphasizing because the management of cerebral palsy is so often based on what is done for disabilities resulting from injury or disease in late childhood or adult life—that is, for those who have developed normal movements before the onset of their disability. It is not uncommon to see physiotherapeutic and other training methods being practised in these cases which, though appropriate for a disability resulting from an accident or disease that occurred some time after birth, are useless when applied as a routine in cerebral palsy. The treatment and management of cerebral palsy should now be based on what we know of its cause—i.e., prenatal and perinatal brain damage interfering with the development of normal movement.

If the best results in "habilitating" a spastic are to be obtained, treatment should begin in infancy. If this is done, and if the child's mental condition is about normal, the task to be faced when the child grows up will be much less difficult. Every effort should be made to diagnose and treat these cases before the age of 6 months. With the increasing attention now given to cerebral palsy, most children suffering from it will have had some treatment, but even so, the methods adopted are so diverse and so variably applied that when these children reach 16—the school-leaving age in this country—few have as good movement as they should have, and most of them will improve under correct management.

Moreover, the management of cerebral palsied children and young adolescents must not only be correct but must be maintained all day and every day, or many will rapidly retrogress—a fact that is often overlooked.

Movements in these patients require much more muscular effort than in normal people, so every endeavour must be made to reduce to a minimum the physical effort necessary for the particular form of training. Thus, the patient should sit upright on a chair of suitable height and the table or bench should be at the correct height to enable the patient to work with the least effort. The patient should not stand if he can work just as well sitting, for the additional effort of

standing needlessly means wasted effort.

Supervision should be exercised by someone with the necessary knowledge and experience—a doctor or a physiotherapist or an occupational therapist with experience of cerebral palsy cases—during instruction, during meals and at other times, particularly when the patients are trying to do various movements. This is emphasised because so often patients are dealt with adequately in the physiotherapy department but are not so adequately supervised outside it. It follows that for a patient to attend a physiotherapy department for a week or a fortnight is of very little value in itself.

In considering the type of work to be given to a patient, his condition and the extent of his disability must be the guiding factors. The case may be mild or severe or very severely handicapped. Mental retardation may be present or the mental state may be normal or above normal. Slight cases with normal mentality will usually improve and find work which they can do. It is therefore mainly the severely affected who need habilitation.

Bad Habits

There are usually certain defects which must be considered before embarking on the habilitation of any "spastic." Some of these are probably a result of not being properly handled as a child, and consequently the adult patient may exhibit signs of his cerebral palsy which should have been controlled before he grew up. Such defects are drooling, not being able to feed or to dress themselves, and so on. These defects may be the result of overprotection on the part of the parent or guardian and they naturally produce an attitude of mind in which the patient constantly expects to be waited on and makes no effort to do things for himself. It should be one of our first tasks to train such people to do as much as possible for themselves and we should make them realize that these defects make them unacceptable socially. Once they realize how much they can do for themselves they are usually most anxious to try. If they are not allowed to do so they will develop a sense of frustration.

A patient who drools should be taught to swallow by the speech therapist and should be watched and advised at mealtimes until he has overcome his difficulty. He must of course also have his attention drawn to his drooling if he does this at other times.

Feeding.—The patient who appears capable of feeding himself, however awkwardly, should be made to do so, but it may be necessary to teach him the correct movements for feeding himself, or to modify his cutlery so that he can hold a spoon more easily, or arrange for his plate to be fixed. Difficulty in drinking from a cup or other vessel may be overcome by the use of polythene tubing.

Dressing and Undressing. — Patients should be encouraged to do as much of these as they can. Their clothes should be made easy to manipulate; thus, zipp fasteners may be advisable in place ot buttons, on clothes or on slippers or soft shoes.

Bathing and Other Activities. — The chief difficulty that these people encounter in bathing themselves is often getting into and out of the bath. If this can be overcome they can manage the rest. It may be advisable to provide shallow baths, even let into the floor, but if the patient has to be bathed by the staff it means a lot of bending down and heavy lifting. It is usually better to use an ordinary bath, shallow if possible, and either have a hoist by which the patient can be lowered into or raised out of the bath—this is of most help in the very severely handicapped who can do little for themselves—or to let the patient transfer themselves from their wheel chair to a special board put across the bath. The board extends some dist-

ance beyond the bath on the free side, and this end is supported by a leg (as devised by Dr. J. B. Stewart). Instead of a bath, a special chair on which the patient can sit securely will enable him to be well bathed under a shower-bath. Many prefer this, as they feel safer in the chair than in a bath. This chair can be so made that it can also be used for personal hygiene purposes.

Where necessary, hand-rails securely fixed to walls, or other form of firm appliance which the patient can use to pull himself or herself up out of the wheel chair, should be provided, for the more they have to do these actions the more easily they can do them and the more independent they become. The rooms as well as the utensils and implements they use, should be devised with the prime object of providing facilities so that they can do things for themselves.

The chief difficulty in encouraging this is often the staff's feeling that if they do things for the spastic it takes so much less time. That is of course true, but the object is in the long run to ease the work of the staff and make the patients more independent. Once these patients have been trained to be independent they will give much less trouble to their relatives at home, and in some cases these may obviate the necessity of admitting them to homes or institutions.

Breathing.—Apart from these examples of habilitation there is another which, though not strictly coming under this heading, is very important and to which special attention should be directed—namely breathing. Every severely handicapped case of cerebral palsy will show poor or very deficient respiratory movements. These people are much more liable to chest complications in consequence. This also accounts in some measure for their speech difficulties. Proper instruction in correct breathing and its daily practice is one of the forms of physiotherapy that should never be neglected.

Chewing and Swallowing. — Speech therapy which teaches the correct movements of the lips, tongue, cheeks, larynx and chest is an important form of treatment. Instruction in chewing and swallowing should be combined with this where necessary. When food is properly chewed and swallowed it will help normal growth. Cerebral palsy patients are often underdeveloped, partly as a result of lack of proper nourishment owing to faulty chewing or swallowing. If these points seem to be stressed unduly think how often adult spastics are seen dribbling from the mouth—a horrible and wholly preventable sight. Similarly, how many adult spastics eat their food in a decent manner?

Social Contacts.—All these efforts aimed at producing a normal appearance should be encouraged and stimulated by as many social contacts with ordinary people as possible. This means a stimulus to paying attention to dress and appearance. Cleanliness particularly should be stressed and women encouraged to make the most of their appearance by the use of cosmetics.

In our efforts to develop social contacts a good deal can be done by making the patients mobile. This can be done by the use of tricycles, hand-propelled chairs or electric motor-chairs. The fact that a spastic requires a walking aid should not lead us to think that he cannot be trained to ride a tricycle. It has been surprising more than once to find a very severe athetoid learning to ride a tricycle well. This not only encourages the development of correct movement of the legs, arms and body, but also, besides helping in making contacts with ordinary people, develops a sense of independence and responsibility.

At Ponds Home no restrictions are placed on patients going out walking or on tricycles or in chairs except that they must let those in charge know where they are going. A consequence of this freedom is that they are accepted by the village as normal people and invited to all local functions.

Furniture.—I mentioned earlier the importance of patients being able to transfer themselves from a wheel-chair to an ordinary chair and it is wise to insist on the use of ordinary furniture and not ask for special chairs and tables. Such furniture can be adjusted or modified to suit the individual patient. The use of ordinary furniture helps the patient's family and other people they may be visiting, and lays emphisis on normality rather than on handicap.

Physiotherapy

No set scheme of physiotherapy can be laid down that will be suitable for all cases of cerebral palsy. Every case must be treated individually. Various types of physiotherapy have been and are advocated in these cases and most will produce some improvement provided the patient's mental capacity is normal or nearly so. But basically all these methods produce their result by bringing the patient some of those varied stimuli by which the normal child develops his mental picture of movement—stimuli of which the cerebral palsied have been largely deprived because of their brain damage and the consequent diminution of their capacity for developing.

But the choice and correct use of physiotherapeutic methods also depends on a correct diagnosis of the type of cerebral palsy present. We must also recognise the fact that in a spastic case too forcible movements will increase the spasticity and therefore make the particular movement more difficult. Reference is often made in these cases to the term "relaxation". True relaxation of a spastic muscle cannot be obtained thus, and it is therefore a mistake to talk of bringing about relaxation in a case of spasticity. On the other hand, in the physiotherapy of athetosis the teaching of relaxation is an essential factor in the management of the case. If relaxation can be obtained in such cases it will be possible to progress to passive movements and later to active movements.

Other physiotherapeutic methods, such as massage or patting the skin, are of little general practical value. Indeed, it is difficult to understand how massage is going to help a muscle whose condition is due to a brain injury and not to any damage to the muscle, but it may prove useful in a few cases.

Experience does show that the severely handicapped cases of spastic cerebral palsy suffer from a poor circulation, particularly in the lower extremities, and consequently often suffer from chilblains, and heat in the form of hot leg baths or lamps will help.

Relaxants

These might be expected to help by making voluntary movements a little easier in spastics and by reducing extraneous movements in athetoids, but in practice their effect appears to be negligible and they have been abandoned by most people of experience though frimidone has proved valuable in some cases of tension athetoid cerebral palsy.

Details of Training

These, then, are some of the first essential practices in "habilitation". Now I want to offer some observations on the more particular and diverse forms of training. It is difficult to consider these in relation to the various categories into which cerebral palsy is frequently classified—monoplegias, diplegias, etc.—for every case must be considered individually. But there is one exception to this —a hemiplegic whose history suggests a brain lesion after birth and in whom one side of the body functions normally. Such patients seem more suitable for training as "handicapped" persons than as cases of cerebral palsy. They generally find work and unless they have another disability, such as loss of speech, manage to maintain themselves. If the more severely affected are to do a remunerative job they should be able to read, write or type, and should have had some general education.

Education.—Some patients arrive at adolescence without being adequately educated, and they should therefore be instructed in suitable subjects. This may need ingenuity in devising a suitable method of imparting the knowledge. It is desirable, because education enables the adult to lead a fuller mental life. But in relation to providing an occupation for a spastic, it is better to be realistic. A patient may be of high intelligence, but, if he cannot convey his thoughts easily to paper, or to other people, he is unlikely to make a living out of it. Owing to his physical disability this usually precludes the spastic from doing much beyond writing occasional articles or books. In such cases it is probably more helpful to try to find an additional occupation for him.

Selection of Work

In considering what work these patients can do, even if they are very severely handicapped, it is best to proceed on the assumption that the great majority can do something—in fact, generally, more than would at first sight appear possible. Our task is to find out what they can do and bring it to them. In seeking for a suitable occupation, the patient should be consulted and may suggest something one has not thought of—e.g. a very severe athetoid young woman who could do little for herself, and for whom efforts were being made to find an occupation, suggested that she might be a speech therapist, though it is unlikely that she could be so trained. She now carries out the instructions of the speech therapist in the training of other patients—incidentally, with considerable improvement in her own speech, as well as her pupils.

We must try to select work which will not be too difficult, will interest the patient, and, to begin with, will not involve fine movements. All the movements the patient has to do at his work should be regarded as part of his physiotherapy, and as in our normal development we develop the coarser and larger movements first, so should we seek initially for work which provides this. Repetitive work will in time become easier, but in all work we must see that the patient is shown how to do it and tries to perform the movement in a normal manner. The work should not be regarded as an amusement or an occupation to fill in time, but as genuine work. Therefore the hours of work should be fixed and patients should be paid for their work. Nor should repetitive work be regarded as being unsuitable—a great proportion of the ordinary population earn their livelihood by such work.

Where the employment involves much movement it may be wise to break up the periods of work by varying it so as to avoid too much fatigue. Cerebral palsy cases work much slower than normal people and cannot attain their speed if put into competition with them. They therefore generally fail if they are put into an ordinary workshop or factory. It would be wise to accept the fact that only a very limited number are likely to be suitable to work in the general run of works or factories, because the average rate of output is likely to be too rapid. But since they can do similar work at their own speed we should aim at bringing the work to the patient, either in his own home or in sheltered workshops, for the manufacturer who will pay for it. In this way the spastic may not earn as much as an ordinary worker but may earn sufficient to support himself.

Types of Work.—Nowadays there are factories which will send out work to be done at home, such as sewing buttons on cards, making up artificial jewellery, plaster modelling and colouring, pottery for painting, painting toy solders, etc. In addition there are of course the staple occupations of rug-making, basket-work, weaving, stool-making, printing, and many others practised by occupational therapists. Apart from these there are other occupations which do not involve speed and which are quite suitable for

even severely handicapped cases. Poultry farming is eminently one of these. Possibly other outdoor occupations such as pig-keeping, might be tried, but few have experience of this. Certain farming occupations, such as driving a tractor, can be done by the more mobile, and soft-fruit picking and the preparation of boxes of seedlings can be tried. Experience shows that these people usually cannot manage such work as digging or the use of mechanical farming implements such as rotacultivators, nor can they manage motor-mowers—the speed is too fast.

Where they can ride tricycles or use hand-propelled or electrically driven chairs they can be employed in selling or delivering papers or goods, or even running their own shop. At Ponds Home there are three young men who deliver papers, one of whom also does a round selling soap-powders, soaps, and so on.

For women other forms of work are available. Most women want to learn cooking and how to run a kitchen, and they can be trained to this provided we plan the kitchen so that they can get the utensils they require easily and provided the tables, sinks, cooker, etc. are at a suitable height to be worked from a wheelchair. They can also do sewing, knitting, weaving, rug-making, basket-work, etc. The use of special knitting machines enables them to turn out articles more quickly and is therefore more profitable.

Use of Machinery.—The great handicap for which all cerebral palsy cases suffer is lack of speed. Therefore, occupations in which speed is not essential have to be sought, but there is an alternative, and that is to find some way of providing speed for the main or heavy part of the work, and this can be done to some extent by using machinery. The blind are taught to turn out nuts, screws, bolts, etc. by self-regulating machines, and why should not this principle be applied in dealing with spastics?

Machines can be used for woodwork and metal-work, and this vastly expands the varieties of work available.

At first sight, one might hesitate to suggest the use of machines, but it is astonishing and revealing to see how often a patient with active and gross movements can use them, but this probably is where the greatest advance in work-production can be made. As an example, watch a spastic driving an electric chair with one hand, where very slight movements are required, and note his accuracy.

The Mentally Retarded Case.—So far, the suggestions refer to the mentally normal case but with mentally retarded or sub-normal spastics the problem is more difficult. With these people we are unlikely to get as good results in physical effort as with the mentally normal. If, however, their physical handicap is not too great they can be trained to do many of the industrial jobs done by the mentally normal, though the spastic's will be slower and make more mistakes. It is wise, therefore, to try them out in the simpler and easier forms of work, *insisting always on complete accuracy while training.* They can also do outside work, such as poultry-keeping and the simpler handicrafts. They should also be instructed in the various forms of "habilitation" mentioned at the beginning.

Calipers and Splints

Two other factors should be mentioned, but this is treading on very contentious ground, and many people with considerable experience may not agree with the views expressed.

The difficulty that so many spastics have in movement has already been stressed. If, in addition to this strain, their activities are limited by and they have to lift additional weight in the form of calipers or splints, are we not hindering rather than helping them? It is our practice to remove all calipers and splints soon after admission and no case has ever seemed the worse for it. In the majority,

there is more freedom of movement and general easiness afterwards. So far we have not met a single case where, after a trial without calipers or splints, the spastic has wanted them put on again. That is the first point.

The second point is that experience suggests that we lay too much stress on trying to make a spastic walk. Very severe cases can do a lot from a wheelchair, but they should have physiotherapy and be encouraged to make what movements they can. If there is a likelihood of being able to make a patient walk we should do all we can to help. In any case every patient should be encouraged to move his legs as much as possible, but patients are often made to try to walk when they cannot sit up properly or hold their heads up or balance themselves on a chair. Surely we should try to follow the normal development of the infant and concentrate on getting correct movement of the head, arms and body, and balance, before we try to make a patient walk—well knowing that in many cases it is an impossible goal?

Conclusions

In this paper a number of factors have been touched on in this interesting and difficult problem, but it is as well to emphasise that it the *patient* who has to do the work. We can only guide and help—success depends on the patient's own determination and this has to be fostered.

Nearly every cerebral palsy case can do some work, and what they *can* do, they *should* do. We should insist on the fact that they are normal members of the community and therefore have certain responsibilities in the way of trying to support themselves, wholly or in part. Likewise, every effort should be made to encourage those who can work to help their more handicapped companions.

Experience shows that when they realise they can do some work spastics are only too anxious to try, and they are generally most cooperative in trying to help their more handicapped companions.

SUMMARY

In cerebral palsy, brain damage before or at birth interferes with the normal development of movement. Treatment should therefore aim at bringing to the patient those stimuli or sensations by which a mental picture of movement is normally built up, all voluntary movement being mental in origin.

This calls for continuous protracted expert supervision and instruction in correct methods of movement and decides how physiotherapy can best be employed.

In the training of adults with cerebral palsy, social contacts and the development of independence are of primary importance.

Most of these cases can do some work: our task is to find out what kind of work is most suitable for them.

" . . . All diagnoses are provisional formulae designed for action . . . In diagnosis as I conceive it there are three stages: observation; interpretation; and symbolisation or labelling. Observation of the patient is divisible into two parts: elucidation of the history and the physical examination."—LORD COHEN. *Lancet*, 1943, i, 23.

BOOKS NEW AND NOT SO NEW

The School Health Service

S. LEFF and VERA LEFF. *London: H. K. Lewis.* 1959, *pp.* 316, 30*s.*

The School Health Service recently completed its first half century, so this is a timely review of its origin, development and functions, with suggestions for improvements and future work.

If one compares the conditions of the schools and the children at the beginning of this period and now, one must be struck by the remarkable awakening of this nation's social conscience and the vast changes that have taken place in a relatively brief time.

Much of the change has been effected through the devoted service of local authorities and the members of the school health team—teachers, doctors, nurses and welfare workers. No-one reading the book could doubt the need for the School Health Service or deny the authors' plea for better planning and coordination with the country's general health service. We have come a long way in removing the chief cause of physical ill-health, but we still have almost as far to go if we are to achieve positive, abundant, physical and mental health. The School Health Service must help the young towards healthy citizenship.

In successive chapters this book deals with the birth of the School Health Service, its early growth, the challenges of the first world war, the intervening periods, the second world war, and the post-war period. The story shows how lessons were learnt only to be forgotten, and how the problems and experiences of war resulted in better nutrition and a determination to improve standards all round. We see the severe malnutrition which existed among the nation's children, and the value of school meals and milk, vitamins and balanced national diet, supplied under the stress of war. Hygiene, housing, sanitation and proper food have largely removed the disgraceful amount of ill health and wiped out the crippling and killing diseases which afflicted British children.

Having filled in the background, the authors discuss the problems of treatment, which they rightly link with medical inspection and the organisation of the general health service. The Education Act of 1944 for the first time provided "free, full treatment of schoolchildren" other than that given at home. It did little to coordinate treatment, remove overlapping or close the gaps.

Further chapters discuss the part of physical training, health education and nursery schools in promoting good health, once good food and good environment have removed the major cause of physical ill health. The aims of the Service have changed—old functions have become relatively superfluous, while other needs are being realised. In the special environment of the school, opportunities exist for promoting health in ways that could not be attempted while the bad conditions prevailed. This work can only be done if there is proper coordination with the General Practitioner Service, before, during and immediately after school life. In addition, there must be closer coordination with the Social Services, particularly the Youth Employment Service, the factory doctors, and special services for the disabled.

Many criticisms have been levelled at the efficiency of school medical examinations. Some are undoubtedly valid. Many criticisms could justly be made of all the medical services of the country, whether general practitioner or specialist. There is

scope all round for improving facilities for work, coordinating services, and improving the system and the use of records.

This book is a factual survey, so it says very little about changing conceptions of health and the importance of the mental aspect, which must in the long run profoundly affect the whole practice and organisation of medicine. Over and above the health service for the average child there remains the urgent problem of services for the small proportion of children who have afflictions beyond the range of general remedies. Here again, much has been achieved but much requires to be done. For the maladjusted there are child-guidance clinics, but there are also long waiting-lists and inadequate facilities. In some areas there are no clinics at all. The authors rightly stress the need for close observation in school to discover potential trouble at the earliest possible moment. The doctor has an important place to fill in case conferences. Medical students need much more training in mental health, and practitioners often require post-graduate courses to enable them to cooperate fully. The surprisingly high figure of 8% is mentioned as the proportion of children who require psychological aid at any one time, but this figure includes those with asthma, eneurosis and speech defects, all of whom require some psychological assistance.

The problem of rheumatic diseases and tuberculosis is discussed, but only later in the book is reference made to the astonishing decline in the incidence of both, largely since the end of the last war.

The problem of the mentally handicapped might well have been discussed at greater length, particularly since many of the physically handicapped also have some mental retardation.

The importance of family relationships is rightly urged, and the need for early ascertainment of mental as well as physical defect. Children who fall behind in education because of some unrecognisable handicap are soon caught in the vicious circle where backwardness reacts on health and health upon learning. The authors underline the need to make life as normal as possible for such children, only sending them to special schools if they need special techniques and if their training is beyond the capacity of teachers in ordinary schools. The extent of the child's handicap often depends not primarily on his physical defects as on personal attitudes and the influence of family relationships and social environment. Defects must be overcome by compensating training of other faculties. The defective child must be judged by what he has rather than what he lacks. Only one in a thousand, say the authors, is so handicapped that he can never support himself or lead a full life. Institutional treatment is in most cases very expensive, ineffective, or both, and more emphasis might have been placed on the large number of mentally and physically handicapped children who are capable of training for useful employment. It is encouraging to know that the Minister of Health intends to call on local authorities to submit plans to deal with these problems.

The stigma of mental defect must be removed. Mind and body, physical and mental, are part of a single entity. The projected hostel and training centre at Slough, where there will be facilities for a sheltered workshop, hostel life while working in industry, or a training followed by full life in the community, may well be the forerunner of many more such schemes. A particular feature of the arrangements will be that physically and mentally handicapped—and many are both—will be in adjacent centres, and the more able of the physically handicapped can take an important part in supervising and training the mentally handicapped.

A great deal has been done to improve the health of the nation's children. Never before have such fit and happy children emerged from our schools to face the adult world. No longer are the less fortunate

segregated and forgotten; wherever possible they are kept in ordinary classes in ordinary schools or special classes in ordinary schools. If special classes are necessary, every attempt it made to fit their pupils for return to an ordinary school.

There is still a lot to be done—we need better facilities for special training, with smaller classes and more teachers, as well as improved techniques in training. Much remains to be done to educate the public in the nature of handicaps and the need to accept the handicapped into community life. The public must realise how much the handicapped can do, with adequate training, to support themselves. The School Health Service must link up with the Maternity and Child Welfare Service and follow through to the end of adolescence in cooperation with the general practitioners and the rest of the National Health Services.

This book is excellent for reference and a guide and encouragement for the future.

M. E. M. HERFORD.

Adolescence to Maturity

By V. C. CHAMBERLAIN. *London*: Penguin Books. 1959, *pp.* 94, 2*s.* 6*d.*

Mr. Chamberlain himself subtitles his book "A Practical Guide to Personal Development, Fulfilment and Maturity". He deals with the social, emotional and philosophical aspects of maturity with regard to careers and leisure, courtship and marriage, and concludes with various recognised modern attitudes to life; and with the learning of the "art of living". His own background, he explains is an industrial apprenticeship, followed by social science studies at Reading and Manchester Universities; and then educational work, especially among school-leavers and young workpeople.

This handbook, first published in 1952, has been written as an introductory textbook for the teenager embarking on life and adult status, stressing the need for social adaptation with broad interest and a balanced healthy outlook supported by an adequate philosophy. This philosophy of life, says Mr. Chamberlain, is the "architectonic framework of the mature personality", making for orientation and integration. He gives us some practical advice on work and leisure and deals at length with the practical and "religious" aspects of sex, love and marriage. In his chapter on "Attitudes to Life", he briefly defines what he considers are the scientific, ethical, aesthetic, practical and religious attitudes to life and their practical applications. His final chapter, "Some Hints on the Art of Living", is a general survey of the qualities which, in his opinion, make for maturity and fulfilment.

The author's straightforward non-literary style, combined with clear headings and layout, makes his book extremely readable. He writes concisely and simply, with good illustrations, and the impression conveyed is one of sincerity. He is broadminded but not afraid to grasp the nettle. His advice is sound, up-to-date and practical, and as an experienced social scientist he is well acquainted with his subject. He seems to have tackled rather too wide a field for the small volume that the Penguin series demands. His attempt at a comprehensive treatment of adolescent problems often results in too general a picture. He cannot do more than summarize many of the facets of the subject, and when he tries to go into detail, as in the chapter on love, sex and marriage, he throws the rest of the book out of balance by emphasising its superficiality. Conversely, in his attempt to discuss the religious attitude, his brevity leaves the impression that he has little conception of Christianity.

The book will meet the needs of many young people who could not digest a more profound exposition, and if Mr. Chamberlain's many constructive sugges-

tions lead his readers on to further study, he will have done a valuable service. Its attractive new form, arresting cover, and handy size should ensure the book a wide sale.

MARGARET A. SMITH.

Mental Retardation

Its Care, Treatment and Physiological Base. By HANS MAUTNER, M.D. *London*: Pergamon Press. 1959, *pp.* 260, 35*s.*

This is a collection of 25 lectures, not a textbook. It is apparently written from the standpoint of certified inpatient practice in a large New England State mental deficiency hospital. To the English reader this seems to bias the selection of the cases described, and, despite an introductory chapter, it is not clear what is meant by mental retardation. The second and third chapters are on the autonomic system, describing trials at the author's hospital and treatment with psychotomimetics and tranquillizers. Unfortunately, official and trade names are used indiscriminately, and the tangle of eulogistic papers published in this field is reported uncritically and without much order. It is, therefore, not possible to decide which type of patient is likely to benefit from which drug. Later chapters discuss aetiology, noting as important points: that the moron group is to a high degree genetically, rather than environmentally, determined; the inherent "criminality" of defectives, the latter term remaining undefined; that children of defectives or criminals, "grow up in institutions" much better than in their parents' care; the Kallikak family; Lombroso's "degenerative signs" of criminality and, by inference, of defectives; a list of clinical findings presumably indicative of familial oligophrenia under a chapter heading of this name. It seems a pity to find these statements in a book published in 1959 when so much work has recently been done relegating them to their proper place.

Ensuing chapters include accounts of neurological diseases such as Friedreich's, bone diseases such as achondroplasia, and lipodystrophies such as Gaucher's disease. These are not usually nursed in British mental deficiency hospitals. There is also a detailed account of several neurological syndromes in which the author says, and one has to agree, that mental retardation never occurs.

The interesting chapters on traumatic factors in the aetiology of defectiveness do not mention the outstanding results of Pasamamick, Lilienfeld and others correlating prematurity and pregnancy toxaemia with defectiveness, but give a full acount of accidents in the home, which rarely cause defect.

The fifteen-page chapter on cerebral palsy is concerned entirely with drugs, and does not mention physiotherapy or surgery.

A short chapter on psychoses considers schizophrenia as one cause of defectiveness, but since the only treatment discussed is psychoanalysis little hope is offered. The only mention of other mental disorders, such as psychopathy or personality disorder, which are often found in defectives, is a paragraph on sterilization. "Direct treatment of mentally retarded people is . . . much more difficult", the book concludes, "and very little real sucess has been achieved". The author ends by noting that the "treatment of very low grade 'vegetables' is hopeless".

Dr. Mautner, who seems to be principally interested in drug treatments but defers at times to the psychoanalytically orientated environment, gives a timely reminder of the dangers of working only among inpatients. Although mental deficiency is defined legally in both the U.S.A. and Britain, practice differs widely, particularly with high-grade defectives, and it is never clear whether or not the author is referring only to certified inpatients. Since his terms are

not defined, it is of little help to take large heterogeneous groups of defectives, submit them to drug-treatment without controls, and record improvement. Sound conclusions can seldom be drawn under these circumstances.

Two of the book's valuable features are its description of current German work and its many references. It is not clear whether it is intended for nurses or psychiatrists; 1,100 references are too many for the former, yet many simple paragraphs are presumably not intended for the latter. It is well printed, neatly bound and attractively presented.

M. J. CRAFT.

ABSTRACTS

IN COLLABORATION WITH *"Abstracts of World Medicine,"* PUBLISHED BY THE BRITISH MEDICAL ASSOCIATION

The Natural Clinical History of Choreo-athetoid "Cerebral Palsy"

P. E. POLANI. *Guy's Hospital Reports* 1959 **108**, 32-45. 1 fig., 31 refs.

The author studied 73 patients with choreo-athetoid movements accompanying "cerebral palsy" (defined as "a persisting qualitative motor disorder appearing before the age of 3 years, due to a non-progressive interference with development of the brain") in order to obtain a clinical picture of the evolution of the neurological features.

In 38 cases the condition followed severe neonatal jaundice, due in 22 cases to Rh iso-immunisation; in 35 no history of jaundice or of disturbance of pregnancy was elicited. The family histories were all non-contributary. There was a high incidence of prematurity in the cases not associated with Rh iso-immunization. In the group with no history of neonatal jaundice there was a high incidence of abnormalities of labour and of neonatal disturbance of feeding and oxygenation, neonatal fits, and vomiting. In most of the 73 cases neonatal symptoms had disappeared by the end of the second week of life, but feeding difficulties and respiratory stridor often persisted.

After the second month of life in most cases some neurological disturbance became evident. (1) In 8 cases this merely took the form of delay in development. (2) In 37 cases there were opisthotonic attacks, between which (with the exception of one case) the child was "floppy". The attacks became more frequent and severe, until about the age of 9 to 11 months after which they often decreased in frequency and severity, being followed in about half the cases by a short phase of hypotonia and in the remainder by "unwanted" movements of the limbs. (3) In 25 cases the picture was of hypertonus, with relaxation only during sleep or at rare intervals during the day. This usually gave way to unwanted movements. (4) In 3 cases there was striking hypotonia with developmental retardation. By the end of the first year hypotonia was the main clinical feature in at least 23 cases, while others were hypertonic or exhibited only developmental retardation. Of the 56 cases in which the age of onset of unwanted movements could be estimated with reasonable accuracy, they appeared before the age of one year in 2, before 2½ years in 39, and between 3 and 3½ years in 17. The predictive value of the early findings in cerebral palsy is discussed.

R. Wyburn-Mason

Survivorship in Cerebral Palsy

E. R. SCHLESINGER, N. C. ALLAWAY, and S. PELTIN. *American Journal of Public Health* March, 1959, **49**, 343-349, 8 refs.

Surveys of the prevalence of cerebral palsy show that the rate diminishes with increasing age after adolescence. In Schenectady County, New York, it was found in 1948 that the prevalence rates for the age groups 5 to 9 and 10 to 14 years were 4·4 and 2·5 per 1,000 respectively, whereas in the age group 25 to 34 years the rate was only 0·8 per 1,000 while over that age it was 0·1 per 1,000. Accurate knowledge of survivorship in cerebral palsy is limited, but estimates based on clinical impressions have placed the mortality in the first 5 years of life as high as 15% compared with a rate of less than 5% in the general population.

During the 3 years 1950-2 compulsory notification of all cases of cerebral palsy was in effect in New York State (exclusive of New York City). Details were thus obtained of 3,299 individuals under the age of 18, from whom the 3,108 who were born before January 1, 1950, were selected for the present study of survivorship. During the 7½-year period of this study 119 deaths occurred among the 1,708 males and 86 among the 1.400 females. The over-all death rate for males was 9·6 per 1,000 person-years and for females 8·5 per 1,000 person-years. The male mortality was found to be 13 times and the female mortality 17 times greater than that to be expected in a general population of similar age distribution.

In 2,586 of the 3,108 cases the extent of the physical limitations was reported, and a study of mortality in this group provided further information. There was a marked increase both in the observed death rate and in the ratio of observed to expected deaths in each sex with increasing severity of physical disability. The mortality among those with severe physical involvement was 27 to 30 times greater than that expected in the corresponding age and sex groups of the population at large, while among those with mild physical involvement the mortality rate was 4 to 5 times greater than expected. No attempt was made to assess mortality according to the type of involvement since few patients had only one type, spasticity being combined in many cases with athetosis, ataxia, or rigidity. However, 15% of the cases were reported originally from state institutions for the mentally retarded, and the mortality in this group was 30 times greater than that of similar age groups in the general population. This excessive mortality was probably due to the high proportion of cases of severe physical involvement in this group as compared with the series as a whole.

J. Browne Kutschbach

The Startle Reaction of the Infant

S. WIESER and K. DOMANOWSKY. *Archiv für Psychiatrie und Nervenkrankheiten* 1959. **198**, 257-266, 4 figs., 13 refs.

The so-called Moro or "startle" reflex—the reaction of the newborn child to a sudden sharp blow on the mattress on which it is lying—was analysed by means of high-speed cinematography (400 to 600 frames per second). The reflex was found to consist of 2 phases, the first being a general convulsive flexion of the body. The second phase is a complicated reaction based on various interrelated vestibular and postural reflexes, starting with a turning movement of the head, which is followed by rotation of the shoulder and upper part of the trunk and finally by flexion at the hips and knees and dorsiflexion and supination of the foot as if in preparation for jumping.

W. Mayer-Gross.

The Development and Pathology of the Startle Reaction

S. WIESER and K. DOMANOWSKY. *Archiv für Psychiatrie und Nervenkrankheiten* 1959. **198**, 267-273, 10 refs.

The authors, using high-speed photographic methods [see Abstract 413] have made an analysis of the normal and pathological development of the startle reflex during the first year of life. The flexion reaction of the first phase becomes confined more and more to the face and neck, while the acoustic stimulus becomes dominant for the release of the reflex. In diseases of the extrapyramidal motor system, however, the generalized flexion may reappear as a sign of regression. The second phase disappears after the third month, the various reflexes being disintegrated or incorporated into higher mechanisms of behaviour.

W. Mayer-Gross.

Lesions of the Temporal Lobes Complicating Infantile Cerebral Palsy

J. B. CAVANAGH. *Guy's Hospital Reports* 1959. **108**, 21-31, 5 figs., 17 refs.

In this study reported from Guy's Hospital Medical School, London, the author has attempted to discern the sequence of events which may lead to damage to the temporal lobe of the brain in consequence of local cerebral injury from any cause elsewhere in the brain, with particular reference to 4 cases of mild infantile hemiplegia drawn from a study of more than 50 temporal lobes removed at operation for the relief of temporal-lobe epilepsy; some of these have been previously described (for example, by the author and Meyer (*Brit. med. J.*, 1956, 2, 1403; *Abstr. Wld Med.*, 1957, **21**, 280). In only one of the 4 cases was there severe mental retardation.

In 3 of these 4 cases the temporal lobe lesions consisted of the well-known disseminated loss of nerve cells in the pyramidal layer of Ammon's horn, the uncus, and to a lesser extent the second and third layers of the cortex, especially in the anterior and infero-medial regions and in the depths of the sulci. In about half of the 50 cases forming the whole series there was loss of nerve cells in the basal nuclei of the amygdaloid complex. There was usually a variable degree of gliosis.

These abnormalities are met with in a variety of states of cerebral disturbance, for example, irreversible hypoglycaemia coma, and have been attributed by some authorities to herniation of the region through the tentorial opening during the moulding of the head at birth; yet a history of birth trauma is not forthcoming in many cases and the onset of cerebral disturbance and epilepsy occurs suddenly in early childhood, often following a febrile illness or teething troubles. Other authors have ascribed the changes in the temporal lobe to vaso-spasm associated with serial epileptic convulsions, combined possibly with a special type of "consumptive anoxia" induced by the excessive metabolic work of certain cells during these seizures, but in the present author's opinion these older views are too vague to gain general acceptance. More recently Lindenberg has suggested that the simple mechanical effects of oedema may be largely responsible for the changes. The selected regions involved are those supplied by long penetrating arteries, which are readily susceptible to compression or stretching over prominences induced by the oedema.

R. Wyburn-Mason

Cerebral Pathology in the Newborn

A. E. CLAIREAUX. *Guy's Hospital Reports* 1959. **108**, 2-20, 5 figs., 40 refs.

The author reports, from Queen Charlotte's Maternity Hospital, London, a post-mortem study of 410 newborn infants and 646 still born foetuses weighing over 500 g. Of these two groups an intracranial lesion was found in 168 (40%) and 147 (23%) respectively and was the cause of death in 118 of the former group and 117 of the latter. In the two groups taken together the most frequent cerebral lesion was one due to birth trauma, which was present in 120 cases. The usual lesion from this cause is laceration of the tentorium on one or both sides and less often of the falx cerebri, or rupture of the great cerebral vein of Galen. When birth trauma leads to intracranial haemorrhage the bleeding usually occurs into the subdural space. The next most common intra-cranial lesion was anoxic intracerebral haemorrhage which was present in 89 cases and was the cause of death in 45, being an associated finding in 44. In this condition the haemorrhage may be extensive (intraventricular or subarachnoid) or patchy or petechial. Developmental defects of the brain accounted for 56 of the stillbirths and 23 of the neonatal deaths. The two chief abnormalities were hydrocephalus (34 cases) and anencephalus (27); other defects included cranium bifidum (15 cases, with encephalocoele in 13), inencephalus (2), and microcephalus, hydranencephalus, and cyclops (one each). Kernicterus accounted for 22 of the neonatal deaths, in 15 of which it was a complication of haemolytic disease of the newborn. Lastly there were 3 cases of cerebral infection, 2 being of meningitis and one of toxoplasmosis.

The author discusses the various factors concerned in producing the cerebral changes. In the case of developmental anomalies

these are not only genetic, but also environmental, such as rubella infection of the mother and exposure to low oxygen tension or x rays. The part played by cerebral anoxia in the causation of stillbirths and neonatal death and also of athetosis or cerebral palsy in infants which survive is stressed. In many of these cases the mother gives a history of vaginal bleeding during pregnancy and thus the anoxia causing the cerebral lesion may not have occurred at the time of delivery. The incidence of fatal trauma at birth is declining with improved obstetric care. The questions whether birth trauma is responsible for neurological sequelae in the affected infant and, if so, what form they take remain controversial.

The frequency with which kernicterus occurs in cases of haemolytic disease of the newborn depends on the serum bilirubin concentration; it is rare when this level remains under 20 mg. per 100 ml. Modern methods of treatment of haemolytic disease by exchange transfusion have largely abolished kernicterus in the institutions where this procedure is practised.

R. Wyburn-Mason.

Infantile Spasms and Hypsarrhythmia

B. D. BOWER and P. M. JEAVONS. *Lancet*, March 21, 1959. i, 605-609, 2 figs., 26 refs.

In this clinical and electroencephalographic (EEG) study of infantile spasms (also called lightning spasms, salaam spasms, and many other names) the authors have attempted to establish the differences and degree of correlation between this condition and hypsarrhythmia, as seen in 22 children with these conditions attending the Children's Hospital, Birmingham, during the 3-year period 1955-57. The spasm, which is described in detail, consists in a sudden, brief, generalized myoclonic jerk in which the trunk or neck may either flex or extend and several episodes may be rapidly repeated after a cry. Flexor spasms occurred in 15 cases and extensor spasms in 7; the latter are differentiated from "tonic spasms" by the speed of the attack. (The term "akinetic attack" is avoided because of its lack of precise definition.)

In all but one of the 22 patients the first attack occurred before the age of 10 months, and 21 showed mental and/or motor retardation at their first visit, this being gross in 15 and moderate in 6. The only child with normal mentality at the first consultation became retarded later. Associated neurological lesions were uncommon, and varied considerably. In 8 cases undoubted perinatal cerebral damage may have been an aetiological factor and these patients were retarded before the onset of the spasms, whereas in 11 the neonatal progress was normal and only one of these was retarded before the fits began. The authors state that in this latter group the aetiology is quite obscure. Treatment with anticonvulsant drugs has been disappointing in the hands of all investigators, but the spasms often diminish in frequency and may spontaneously disappear in time. Of 20 surviving patients in this series 19 remain hopelessly retarded—a common experience reported by other workers. Only one patient recovered, after showing moderate mental retardation, and she is apparently normal at 2¼ years of age.

In all but one of these children the EEG recordings were abnormal, but only half showed the chaotic pattern described as hypsarrhythmia; the other half showed a more organized, epileptic, pattern. Comparison with the EEGs of 24 epileptic children showed that only 2 of these exhibited hypsarrhythmia. The authors conclude that a normal EEG pattern is strong evidence against the diagnosis of infantile spasms. No correlation was found between the presence of hypsarrhythmia and the type of the spasm (flexor or extensor), or the degree of mental defect, but perinatal brain damage was more often associated with an EEG record of epileptic type. *John Lorber.*

" . . . Even in the United States, where there is one physician for every 750 people, a recent report to the Secretary of Health, Education and Welfare indicated that a minimum of 14 new medical schools should be built by 1970 to meet the requirements of medical service to a growing population and expanding economy".—RAYMOND B. ALLEN. "Medicine—A Lifelong Study." *British Medical Journal*, 1959, ii, 319.

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